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Clinical Method

A Guide for Medical Students

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Part I. The logic of clinical method

Discard in the first instance all attempts to identify or to name, and try instead to read the malady, tracing the symptoms to the seat of their cause, and discerning the nature of the morbid process by their character and course.

Gowers 1982

Introduction

In the absence of any clearly defined logic of clinical method, the most common road by which you learn (and we teach) is through recognition of patterns of disease. And there is no doubt that this works fairly well for the experienced physician. But it takes time to accumulate knowledge of these patterns, and now that we are introducing you to clinical aspects of medicine much earlier in your course, there is a need to delineate guidelines to help you make clinical assessments, independent of any knowledge about disease patterns. Indeed, I suspect that with the rapid increase in medical knowledge, and the increasing complexity of diseases, some sort of method of seeing the wood from the trees is fast becoming necessary even for the relatively experienced physician. Also, pattern-recognition can never work if one is confronted by a new disease, or disease-variant. And, of course, even with the commoner conditions, each patient is an *individual,* so that there is a real danger in forcing him or her into some pigeonhole of disease, especially if there is any important deviation from its typical form.

Despite all that has been said about clinical logic, it has never actually been defined. It is said that most good physicians work by 'hypothetico-deductive' reasoning, i.e. as clinical data is accumulated during the interview and physical examination, different ideas of their meaning spring to mind and further evidence is then sought, for or against the proposal, which is then either rejected or accepted depending on the outcome. But for the student without much knowledge of disease the real difficulty with this method is: 'Which hypothesis?' Those who have written on this subject suggest that any hypothesis is reasonable to begin with, but time is short, and there must be something more than a random process involved, i.e. some hypotheses must occur to us as more probable than others if this method is to work.

In the absence of a means by which 'good' hypotheses about clinical data can be identified, particularly in the early undergraduate years when medical knowledge is slight, this approach is not particularly valuable. What we have to do is find some guide to *synthesising* clinical data, and what follows is a suggestion of how this may be done, given that you already have, by the pre-clinical years of your course, the basic groundwork of anatomy, physiology, general pathology etc. with which to approach clinical data

The Method.

When embarking on any process of detection, the first questions in one's mind should be those which *group* information into broad categories, rather than those aimed at levels of detail. For example, the question: 'Is there *anything* wrong *at all* with the cardiovascular system?' is a much more important initial question than: 'Does the patient have mitral stenosis?' in the detection of why a particular patient is suffering from shortness of breath.

In general, *we can use our knowledge of anatomy, physiology and general pathology to group information along various lines,* and this is quite capable of getting us very close to a diagnosis even where we do not have any prior knowledge of the various specific disease processes concerned.

There are four important questions which should be considered quite separately when you analyse clinical information, viz: Where, What, How, and Why.

'I keep six honest serving men,
They taught me all I knew.
Their names are What and Why and When,
And How and Where and Who.'

R. Kipling

(1) Anatomical Diagnosis.

. **Where** is the lesion?.

Throughout the history taking and physical examination one should be listening and looking for evidence of which *system* is involved. In the history we often get clues from this not only by our knowledge of anatomy, but from physiology, because disturbed function may give us an important clue to the anatomical system involved; e.g. shortness of breath which gets worse when the patient lies flat is probably due to impairment of left heart function rather than a respiratory cause, because increased pulmonary venous pressure (tending to reduce lung compliance) in early heart failure will be aggravated by lying flat. Similarly the distribution of referred pain can often give us a clue to the *system or organ involved,* provided we know our dermatome innervation, particularly in relation to referred pain. Indeed, neurophysiology is vital for localising the anatomical site of any trouble in the central nervous system.

Actually, our knowledge of function often turns out to be even more important than direct anatomical knowledge in deciding an anatomical diagnosis, especially in the relatively 'hidden' organs such as the nervous system, lungs, heart, and to a certain extent the abdominal organs. In those cases, it is the symptom itself, its *quality, its radiation, aggravating* and *relieving* factors, which often give the most important clues to the anatomical site, system or organ involved in the disease process, as illustrated above.

Physical examination also adds to Anatomical Diagnosis by giving us direct evidence about the organ involved, particularly if we understand the anatomical relationships of the various organs and their surface anatomy, e.g. respiratory signs over the right lower chest anteriorly indicate involvement of the right middle lobe by *some* process. (The precise nature of this process is not considered when addressing this Anatomical category of diagnosis, but separately as indicated below, under Pathological Diagnosis.) Of course physical examination is particularly important in defining the anatomical site of conditions visible to our eyes or palpable to the touch, such as skin and joint disease. But so often, in less accessible systems, examination findings are few, and the history becomes of the utmost importance. Note also that some conditions will not appear to be confined to any one organ-system, raising the possibility that we are dealing with a multi-system disease. (However, see 3 below).

(2). Pathological Diagnosis

What, is the general pathological nature of the lesion, independent of its site?

Here we consider only the general areas of clinical pathology and not specific histopathological processes. Thus we wish to know whether the problem is inflammatory, neoplastic, traumatic degenerative, hereditary, immunological, atrophic, ischaemic, etc. The most important data to answer this question come from the history, namely the **time-intensity relationships of the major symptom** or symptoms involved. It is largely for this reason that it is so important to have a clear and accurate history from the patient, particularly that which relates to the circumstances of *onset* and time-course of *progression* of the symptoms. All good histories should start with a clear account of this.

Hyper-acute conditions

When the onset of the symptoms is *dramatic,* occurring within seconds, it is most usually due to rupture or obstruction of a hollow tube somewhere within the body, for example, a perforated peptic ulcer, a spontaneous pneumothorax (rupture of the lung into the pleural cavity), or an acute coronary artery occlusion. Note that in many organs, such as the brain, heart, etc., the only hollow tubes are vascular ones, and this allows the diagnosis to be narrowed much more than in the case of the abdomen, where the distinction between, say, obstruction of the gut and blood vessels may be more difficult on these grounds alone. Note, too, that in organs where the only hollow tubes are vascular, a sudden onset not only gives us information about Pathology, but also Anatomy, e.g. an acute 'stroke' involving the parietal cortex suggests involvement (probably obstruction) of the *middle cerebra l artery.*

To distinguish between rupture and obstruction of a hollow tube may be difficult, but if the symptoms pass off within minutes, or at the most hours, the condition is very unlikely to be rupture, and far more likely to be (reversible) obstruction such as embolism or arterial spasm. Of course, other acute events include mechanical and traumatic ones.

When a patient has *less acute* onset of symptoms, i.e. occurring over a week or so, the condition is more likely to be inflammatory, toxic, allergic or immunological condition. Again we need to ask broad questions first so as to identify which of these processes is most likely. It is for this reason that we ask about evidence of inflammation, both *general* (shivers and sweats, fever, peripheral blood neutrophilia) and *local* (purulent secretions from the organ concerned), both in the history and on physical examination. Once we know this, we can then go on to further detail about whether it is infective, and if so, whether bacterial or viral or, on the other hand, secondary to necrosis from other cause as in alcoholic 'hepatitis', acute myocardial infarction, pulmonary infarction etc. Note the importance of the question: 'Is there *any* sort of inflammatory disorder?' before considering more detailed questions such as: 'Is this *viral?'* I would emphasise, too, the importance of separating considerations of the pathological nature of the lesion from any analysis of its anatomical site, at least in the first instance, just as Gowers advocated.

Clues about an *allergic* nature to any process will be found in the history, as well as on examination, but if not we may have to go to other investigations, such as serum complement, analysis of blood for immunoglobulins (eg IgE), antigen-antibody complexes etc. *Toxins* (and particularly *drugs)* must be remembered as a potential cause of disease, and a useful clue here apart from the drug-history, is that such agents normally affect the body symmetrically. Thus a peripheral nerve lesion localised to one leg is likely to have a local cause, and unlikely to be primarily toxic as in, say, arsenical poisoning.

Chronic Conditions

That is those lasting months or years. These include the neoplasias, chronic inflammation, chronic immunological disorders, degenerative diseases, and hereditary conditions. Again, clues about chronic inflammatory lesions are often indicated by fever and local purulent secretions, but may also be accompanied by weight loss. Weight loss (and anorexia) in the absence of fever in a *chronic progressive process* over months suggests neoplasia. (But be careful to ask about decrease in appetite, because weight loss despite increased appetite could be due to

increased fuel loss, eg. in the urine in diabetes mellitus, or through the bowel in maldigestion/ malabsorption syndromes; also via increased body metabolism as in thyrotoxicosis).

Remember that what you are first looking for in any category of disease, pathological or otherwise, is the *broad or general* sort of disease process involved, not some specific histo-pathological entity.

In relation to local secretions, enquire not only about evidence of inflammation, but also about the presence of blood or serum, because this could give the clue to a vascular or ulcerative nature of the underlying pathological process.

In determining clinical pathology the history is often of the utmost importance, particularly in relatively 'hidden' organs such as the brain, but also in the cardiovascular system (especially the heart) and to some extent in the respiratory system. Actually, in any of the organ systems where there is an intermittent disorder that you are not lucky enough to observe at the time, the history is the *only* information you have to go on in making a pathological diagnosis.

In more accessible and less evanescent conditions, clinical examination can also provide useful information about pathology. This is most obvious in the skin and joints, where the evidence of inflammation (local heat, redness, swelling, pain and loss of function) and deformity are usually obvious. The same holds true to a lesser extent in at least some of the internal organs, particularly the abdomen, where masses may be palpable to the examining hands. Then, the consistency, shape, size, degree of tenderness and presence of any overlying murmurs, can add usefully to discerning the nature of the pathological process. For example, a grossly enlarged, hard 'knobbly' liver with an irregular surface and edge would suggest secondary carcinoma; a uniformly enlarged firm liver without irregularity would suggest some other *chronic* process (e.g. some condition of infiltrative type, or perhaps cirrhosis in the stage before atrophy has diminished liver size). In sorting this particular situation out, the presence or absence of splenic enlargement is useful (because splenic involvement and therefore splenic enlargement is rare in secondary carcinoma). In other areas of the abdomen, the kidneys may be palpable, and other masses, so that when examining the abdomen, always define not only the anatomical relationships of palpable organs, but their consistency, shape, extent of enlargement, consistency, tenderness, irregularity, and the presence or absence of any vascular bruits, because this will give you important information about the pathological nature of the lesion concerned. Also remember that rectal examination will give you useful information about the macroscopic appearance of the stool, including the presence of any blood, altered or otherwise.

In the respiratory system, you will also sometimes obtain useful information about pathology from your physical examination, including examination of the sputum. *You must always be able to distinguish between consolidation and collapse of the lung, pleural fluid, pneumothorax, and chronic obstructive airways disease* from the clinical signs.

At the extreme, the central nervous system very rarely gives you any direct evidence on clinical examination about the pathological nature of the process involved, (except where there are vascular bruits etc. to be heard). It is in such cases that you are so dependent on a good history for this diagnostic aspect.

After the questions 'where?' and 'what?', we should be in a position to know whether the disease is local or general, anatomically, and if local, which organ or system is involved. We should also have a fairly clear idea of the pathological nature of the process involved, particularly whether it is a *hyper-acute (dramatic onset), acute, sub-acute, or chronic* process. If we have listened to the patient carefully during our history-taking, and collected the information completely and accurately during our clinical examination, we should be able to synthesise the anatomy and pathology of the problem fairly accurately, and hence be very close to a diagnosis indeed For example, in a patient who has had an onset of shortness of breath, with cough, yellow sputum and fever over a 24 hour period, and who on examination has the signs of consolidation over the right lower chest anteriorly, we can say without any prior knowledge of names of diseases that he or she has an acute inflammatory lesion involving the right middle lobe of the lung. (And if he has a high white cell count in the peripheral blood with increased neutrophil polymorphs, it is more likely that this inflammation is bacterial than viral). Thus, without any knowledge of names such as 'pneumonia', etc. we will have come very close to a correct diagnosis already, and merely on a basic knowledge of general anatomy, physiology and pathology. Indeed, 'pneumonia' merely describes an acute inflammation of the lung parenchyma, and the name is only shorthand for what we have found ourselves, nothing more. I would even suggest that you are better off not naming names initially at all, in the same way as Gowers has counselled, since this sometimes carries subtle connotations leading you in wrong directions unconsciously. Thus, the diagnosis 'lobar pneumonia' is most usually due to the organism Strep. pneumoniae, and tends to imply this if you use diagnostic labels without careful thought.

I have no doubt that you will still sometimes fall into the trap of trying to stick labels on patients, regardless of what I have said, so let me say, further, that whenever you do, please analyse each of the syllables within that label, and make sure they are relevant to the *individual patient confronting you (e.g. if* your label is 'acute viral hepatitis', is it really acute, is it really the liver that is primarily involved and, if so, is it truly inflamed, and what is the evidence that the inflammation is due to a virus?)

(3) Functional Diagnosis.

How does the condition affect the patient?

As already noted, in many situations, the functional manifestations, sequelae, and/or complications of a disease process are dominant in allowing us to make our Anatomical diagnosis. In addition, by the time we have addressed this category of diagnosis we usually have most of the clinical information available about the *present* illness to begin to synthesise it to a more complete diagnosis. In this respect, we sometimes have several groups of symptoms that could either be seen as primary causative events, or secondary functional consequences, and this can create difficulties. High blood pressure associated with impairment of renal function is a good example, because hypertension can be either the cause or the effect of the latter. Similarly, paroxysms of tachycardia could either be the cause (low diastolic coronary perfusion pressure) or the effect, of myocardial ischaemia.

When one assembles the data together at the end of the physical examination, unravelling cause and effect in such cases can be very difficult. The important principle here is not just to look at the data itself, but for which symptom came first in time. Clearly, if a patient describes palpitations *first, followed by chest* pain, then it is likely that the palpitations are primary, and

the myocardial ischaemia secondary.

As a general point, you should get used to the idea of stating your anatomical and pathological diagnosis (e.g. acute myocardial infarction) and *following* it with whatever functional consequences are involved in the *individual patient* confronting you, including any *secondary effects, sequelae, and/or complications of his or her disease.* This is the area where traditional textbook diagnoses are so deficient (and of course if you think about it, no textbook or 'off-the-rack' diagnosis could ever describe the functional sequelae or complications in any *individual* patient).

The functional aspects of disease bear not only on the diagnosis but also on the patient's management. The severity of the condition has obvious importance in treatment. For example, a patient who has a very low arterial and central venous pressure with rapid (reflex) tachycardia after a haematemesis needs blood transfusion before any further consideration can be given to other categories of the clinical diagnosis. Similarly in a patient with severe 'acute asthma' and consequent severe central cyanosis, oxygen is needed before we should make any attempts to understand further details of the process involved. The same holds true for all other *emergency* situations. There, we have to act appropriately to treat obvious and severe *functional* disturbances before delving into aspects of the condition of lesser urgency.

Functional consequences or sequelae of disease processes may also have an important bearing on the treatment of chronic conditions, (although there, not usually until other aspects of the diagnosis have been discerned). For example in treating patients with hypertension, the case for anti-hypertensive medication is stronger when there is evidence of its secondary effects such as left ventricular hypertrophy, retinal arteriolar narrowing etc.

(4) Aetiological Diagnosis

WHY did the patient get the condition in the first place?

As you will see in parts II and III of this booklet, what we first aim to do in taking a clinical history and examining the patient is define the nature and anatomical site of the *present illness* confronting us, together with its functional sequelae and/or complications. But once we have answered those questions, we must next ask: 'Why did the patient develop this condition in the first place?' In this regard, if the time-intensity relationships are chronic, we will want to know the long-term **background predisposing factors** which might have been involved (e.g. whether there are the cardiovascular 'risk factors' of cigarette smoking, high blood cholesterol, obesity etc. present in a case of chronic angina due to coronary atherosclerosis). Along the same lines, if the condition is acute, we must enquire about the *precipitating factors,* such as some change in the patient's life-style or some important life-event, exposure to toxins, infectious agent, unusual environment etc. And, of course, when we have an acute-on-chronic process such as chronic coronary atheroma with sudden superimposed thrombosis and consequent heart attack, we will want to find out not just the long-term factors predisposing to the atherosclerosis*, but also the acute factors precipitating the recent event.*

Finding out about predisposing and/or precipitating factors to disease usually comes from the second phase of our history, namely the background to the illness (see Part II of this booklet). You must therefore ask your patient about this background, leaving him initially to tell his own story, and only later asking more specific questions. If you are still at a loss in this respect, always ask the patient what he thought brought the condition on — he may be wrong, but patients often supply interesting and useful insights into their disease process, even if only in a general way.

When you have elucidated the *aetiological* background to the illness as well as its anatomical site, *its pathological nature,* and its *functional* consequences, effects sequelae and/or complications, you should have a complete diagnosis, e.g. acute (pathology) viral (aetiology) hepatitis (liver + inflammation = anatomical + pathological), with secondary impairment of liver function (functional diagnosis). The diagnosis in some other patient might be acute (pathology) myocardial (anatomical) infarction (pathology), with secondary left ventricular impairment and episodic ventricular tachycardia (functional), the acute precipitating factors being uncertain, but the long-term (aetiological) 'risk factors' of cigarette smoking, high blood pressure, and diabetes mellitus.

Synthesising the Complete Diagnosis

Note that once you have identified these various categories of disease they may not always fit together as neatly as the above, but at least now you will only have four sub-conclusions from which to synthesise your diagnosis, and that makes the evidence a lot easier to handle than a whole mass of unprocessed data. Getting the four categories in their correct order and perspective can be difficult at times but remember that what is cause and what is effect can usually be determined by elucidating which came first in time. Remember, too, that some organ diseases can have multiple effects on different systems, so that what may appear to be a multi-system disorder can often turn out to be a single organ problem with secondary manifestations elsewhere. The first principle here (Occam's razor) is to try first to make the simplest diagnosis possible i.e. to see if the condition can be viewed as primarily due to an abnormality of *one* system with secondary manifestations elsewhere (e.g. primary liver impairment with secondary (functional) effects in other organs; primary renal failure with secondary hypertension, oedema, pulmonary congestion etc.).

Sometimes, of course, you will indeed be dealing with a multi-system disorder, such as an autoimmune disease (e.g. systemic lupus erythematosis), but in approaching any information, you should only think of such disorders after having excluded the simpler possibility that the widespread nature of the condition is due to single organ failure with secondary manifestations elsewhere.

The four diagnostic categories are not always mutually exclusive but this can sometimes allow them to *interact* in useful ways. For example, if a patient presents with right hemiparesis affecting the arm more than leg, together with dysphasia, the likelihood is that it is the left parietal cortex involved, anatomically. But if, when we turn to our considerations of pathology, we find that the condition was of sudden onset, (i.e. in our terms, an obstruction or rupture of a hollow tube) then, in the brain, this means that it was vascular, so we can go back to our anatomical diagnosis and extend it by considering it in terms of vascular anatomy, and in the present case this would lead us to the conclusion that the patient had a lesion (probably obstruction through thrombosis or embolism) of the *left middle cerebral artery*.

Secondly, we have already seen how important functional diagnosis can be in localising the

anatomical site of the lesion, particularly in relatively 'remote' organs. For example, in a patient presenting with chest pain not typical of myocardial ischaemia, the finding of impairment of left ventricular function would add some weight to the notion that the pain might indeed be of cardiac origin. In a similar way, the aetiological diagnosis may interact with the diagnosis of the present illness (Anatomical, Pathological and Functional diagnosis). Thus, if the above patient with a typical chest pain and left ventricular impairment was 52 years of age and had a long history of hypertension, cigarette smoking, obesity and diabetes, this would be the right context for a predisposition to vascular disease, and hence would support the general notion that the pain was indeed myocardial ischaemia. On the other hand, if the aetiological background setting was wrong in relation to the preliminary diagnosis of the presenting illness, one would have to rethink the anatomical, pathological and functional aspects of the diagnosis. For example, if the above patient, presenting with chest pain and cardiac enlargement, was aged 15 and had no evident background cardiovascular risk factors predisposing to myocardial infarction, then the whole diagnosis should be re-thought — perhaps he has pericarditis with pain due to that, and apparent rather than real cardiac enlargement due to fluid in the pericardium. These interactions between the various diagnostic categories, though relative, can be very useful in piecing the information together to the best possible final clinical diagnosis.

General Comments

The thrust of this exercise has been to try and give you *guidelines for* making clinical diagnoses without prior knowledge of medical conditions. You may think it slightly odd that we have talked so much about dissecting clinical information before mentioning how it should be collected, but I have done this deliberately so that you may understand the framework in which to collect information. Unless you have some insight into that, your data collection will be unstructured and your diagnoses correspondingly confused. For example, we usually teach that we take all aspects of a clinical history before doing the physical examination, but it must be said that unless we can get a very good idea of the anatomy and pathology of the general problem in the history before we come to the examination, we will not be in a very good position to ask all relevant questions about the background aetiology (e.g. Past History, Family History, Social History, Drug History, etc.) leading up to the condition. In such cases, one often finds that the most important questions one asks in relation to aetiology occur to the consciousness only *after* the physical examination, and hence in practice we often have to go back over the history, looking for factors or events which may have predisposed to, or precipitated, the event. It is for these sorts of reasons that you have to know the structure of the clinical method we use, and the guidelines involved, before you can really go about collecting information to greatest advantage.

Hierarchic Approach to Diagnosis

There is a final important aspect of your approach to collecting information, namely that you ask *broad* questions which allow you to *group* information within the various categories, before moving to finer levels of detail. For example, in a patient with muscular weakness, there is no point in looking for a primary muscular cause if there is good evidence of neurological involvement, e.g. the presence of sensory symptoms and signs. This hierarchic approach of going from broader to more narrow levels allows you to 'corner' the diagnosis at all stages, but it is one of the most difficult aspects of dissecting and synthesising clinical information, and there is no substitute for practice on individual patients to achieve success. No amount of book reading can make you competent in *building* diagnoses for *individual* patients in this way. Another useful hint is to try playing the game of Twenty Questions', i.e. see if you can build up the correct diagnosis in one of your colleague's patients by asking him a series of up to twenty questions. This will force you to ask broader questions before more detailed ones. As an example, when a patient is found unconscious (or when your car suddenly stops), you heed to know rapidly which of the major systems is at fault; (in the case of your patient, is it lack of oxygen supply, lack of blood supply, sudden drop in glucose fuel levels etc.?) In this respect, there is usually one point (at the end-of-the-line, as it were) where each system can quickly be checked, especially in emergencies. For example, in our ailing car, if a bright blue spark crosses the spark-plug gap (end-of-the-ignition system line) when the starter motor is turned over, there is *nothing* wrong with the *ignition system* and the fault must lie elsewhere (? fuel system). If there is no spark, then you have *localised* the trouble to the ignition system. Thus, the first questions must be those where both the answers 'Yes' and 'No' will give useful information. Only when you have localised the system involved by this means should you go on to dissect the problem in more detail. Again, in our unconscious patient, if there is no recordable carotid pulse or blood pressure, you will have localised the problem to the cardiovascular system, and you must then find out whether this drop in blood pressure is due to sudden drop in peripheral arteriolar resistance in the circulation or to a reduction in cardiac output; if the latter, is this due to an altered heart rate or a reduced stroke volume; and if the latter is it that the heart 'can't' (e.g. valve rupture) or 'won't' (e.g. primary heart muscle failure) pump, and so on down the line. This broad, **hierarchic** approach to diagnosis is one I strongly advise you to follow.

When you play your game of Twenty Questions try not to use more, and again heed Kipling's advice:

'But different folks have different views,
I know a person small,
She keeps ten million serving men,
Who get no rest at all.
She sends 'em abroad on her own affairs,
From the second she opens her eyes —
One million Hows,
Two million Wheres,
And seven million Whys!'

From Kipling's 'Just So Stories'.

I ask you to use the method described as a guideline to help you reach diagnoses. As far as possible, try to avoid the pattern-recognition approach, which can be both difficult at your stage of knowledge, and misleading unless you are dealing with anything other than a very classical case — and each patient being an individual means that this rarely happens. As Horace said (65 B.C.): 'If you know any better methods than these, be frank and tell them; if not use these with me'. With this method, you may not always finish up with a tidy diagnosis, but it will usually be a fairly accurate one. And even if you haven't identified the diagnosis totally in all its four

categories, you will at least have 'cornered' it and pointed to the areas or diagnostic categories where more information (e.g. more history and/or investigation) is needed.

An Example of our Approach to Clinical Diagnosis

A 14 year old boy presents with a one-week history of gradually increasing malaise, shivers, lethargy, mild bilateral loin pain, and the passage of decreased amounts of 'smoky' urine, beginning a week or so after the onset of a sore throat. There has been no dysuria (painful mucturition) or other urinary symptoms, and apart from occasional headache, no symptoms relevant to other systems. There is no relevant background past history, family history, social history, psychological history, or drug history.

On examination he has some facial and ankle 'puffiness', a blood pressure of 150/90 mm. Hg., a reduced urinary output, temperature of 38º C., and mild loin tenderness. Urine examination reveals proteinuria (3.0 grams per 24 hours) red cells and red cell casts in the urine, with some of the red cells having unusually fragmented appearances. There are also some white blood cells in the urine.

According to our guidelines, we would construct the diagnosis as follows:

(a) Anatomical site of the lesion

Both history and examination point to the renal system, although we would have to be careful that the high blood pressure was not primary and the renal impairment secondary. However, the blood pressure is only moderately elevated, making the latter unlikely. So, taking the hierarchic approach we have first defined the system involved and now we can try to localise the anatomical problem a little further. The 'smoky' urine suggests the presence of red cells, which is confirmed on examination, and the presence of red cell casts means that the blood has passed through the tubules, so that the bleeding must have come from somewhere in the nephron itself rather than the collecting system (pelvices, ureters, bladder etc.). The renal tenderness is consistent with this conclusion. Just how high up the nephron is the next problem in our hierarchic approach to anatomy. In this respect, odd-shaped red cells usually mean that the cells have been squeezed through damaged glomerular capillaries. Proteinuria of more than 2 grams per day also points to a glomerular site of the lesion (normally less than 0.2 grams per day is filtered by the glomerulus, so this would be the maximum amount we could expect in the urine from any tubular lesion with secondary failure of reabsorption of normally filtered protein). Moreover, there is hypertension, and from our knowledge that renin is released from the juxta-glomerular apparatus in the afferent arteriole this, too, would be consistent with a glomerular site of the lesion. In addition, there is oliguria and fluid retention (oedema) and this could be related to a reduced glomerular filtration rate. All of these features point strongly to the lesion having a *glomerular* site. It is important to stress that we have been led to our *anatomical* diagnosis just as much by *indirect* information relating to derangements in *kidney function* (hypertension, oedema, oliguria, etc.) as by the more direct examination findings (loin tenderness, red cell casts etc.) relating to anatomy per se.

(b) General Pathological nature of the process

Clearly this is an acute process, and probably an inflammatory one in view of the fever and presence of white cells in the urine. Whether this inflammation is due to necrosis, or bacterial or viral infection, or an inflammatory response to allergic or other damage will need more evidence — for example a high *neutrophil* white cell count in the urine and circulating blood would be more in keeping with a bacterial than viral inflammation. An allergic component might be indicated by further history revealing a background tendency to allergies in the past, and this would point the way to a need for such measurements as serum complement, immunoglobulins and other immunological factors in our investigation of the disease.

(c) Functional Diagnosis

HOW does the disease affect the patient?

Our functional diagnosis has already been very important to us in building our anatomical diagnosis. But, as discussed above, it has also been helpful by making us address the question of whether the hypertension has been the cause or effect of the renal disorder. Finally, this diagnostic category will also be important to treatment (e.g. is the degree of impairment of renal function sufficient to warrant dialysis).

(d) Aetiological diagnosis

The question 'Why?' leads us to go back and ask about the background to this illness. In this case, the early sore throat may give a clue, but we might also do well to ask further about allergies etc., in the past.

Without much knowledge of medical conditions, we have therefore built a diagnosis of an acute post-sore-throat glomerulitis (inflammation of the glomeruli) of uncertain cause, with moderate secondary impairment of glomerular function. Actually, once you get to recognise patterns of disease in later years, you will see this as classical 'acute post-streptoccocal glomerulo-nephritis', but you can also see that the two diagnoses are not very far apart, and ours may even be better suited to the individual patient than the 'off-the-rack' textbook label. Thus, our diagnosis includes a statement about the presence and degree of impaired *renal function:* and doesn't assume that nephron segments other than the glomeruli are involved in the patient confronting us, as the textbook label implies? Also, is there really evidence that it is caused by a streptococcus? Again, if you ever do put textbook labels on your patients as you no doubt will do occasionally, at least analyse each syllable, to see whether it does actually fit your *individual* patient, and whether it is complete (especially in its functional and aetiological aspects).

The whole of your pre-clinical learning is designed to help you build up clinical diagnoses in real patients by using your knowledge of physiology, anatomy and general pathology in this very broad and simple way. If you know your anatomy, particularly surface markings of organs, dermatome distributions, neurophysiology, attachments and relationships of the various abdominal organs etc. and if you have trained yourself to *group* information around the four broad categories suggested, you will find this approach not only simple but helpful, even in quite difficult cases, and even where you have little knowledge about the pattern of the disease concerned.

Investigation

Early on in your Clinical Medicine years you will be taught the principles of investigation only. As indicated above, when you are lacking in some diagnostic aspect in an individual

patient, the important further information you must seek first should be more history and/or examination.

The next important principle is that any tests should be done with a purpose and not as a 'routine'.

Merskey's rule: 'Do a silly test and you get a silly answer.'

If you approach clinical diagnosis through the categories suggested, you will soon see where you need to investigate further, because you will recognise which of the various categories is incomplete. Thus, in a patient where you are not sure of the origin of chest pain, an X-ray of the chest may be helpful in elucidating the anatomical diagnosis. On the other hand, when you are fairly certain that the chest pain is anatomically of cardiac origin, but pathology is your difficulty, you may need to take blood for analysis of 'cardiac' enzymes' to help determine whether the episode was a reversible ischaemic one, or an irreversible infarction of the cardiac muscle.

The other important principle is always to do simple non-invasive tests before more invasive ones.

Finally, when faced with having to do very invasive investigations ask yourself whether you are really looking for a cure (i.e. reversibility) or alleviation, because if the latter, your invasive tests are unjustified.

Treatment

The important principle of treatment, which has not changed through the millennia, is: 'First do no harm'. Another principle is to make sure you have the diagnosis right. Then treat all four categories of disease. Always look for reversible conditions, even in situations where they seem unlikely. Also, in these days of increasing specialisation, don't ever forget to treat your patient as a whole. Especially remember that: 'To know what kind of person has a disease is as essential as to know what kind of disease a person has' (Francis Scott Smythe). Think about non-drug treatment, particularly psychological aspects, before leaping in with potentially dangerous drugs. And if the disease outlook is gloomy, at least try to give your patient hope; instil some faith in him and you will be surprised how even patients with incurable illnesses respond.

Part II History-Taking

General approach to the patient:

Be well presented. Introduce yourself. Show that you are friendly. Be alert to elements of the history told in parentheses i.e. as asides, and by nonverbal means of communications such as body language. Avoid appearing hurried. Take only jottings for notes and write the history up more completely subsequently, otherwise you will distract your patient. Be observant while taking a history, particularly observe the patient's general demeanour, including any anxiety etc. and observe carefully his face, eyes, and hands, which often hold a host of clues to the underlying problem**.**

(1) The Present Illness

Patient's account of the current Illness:

Let the patient tell his own story, and get him to do so from the beginning, for example by asking: 'Now please tell me about your trouble from the beginning'. Initially, try not to interrupt, or only do so when the patient starts to get off the track (e.g. by spending a long time telling you what his neighbours thought was the trouble); even then your questions should remain broad and very general, and in eliciting answers, you should avoid asking about specific symptoms at this stage.

Don't lead! Phrase your questions so as to avoid this. If you can only think of yes/no alternatives to ask, then frame the question so as to include both aspects equally, so as to avoid giving the patient any impression that the answer should be 'Yes' E.g.: 'Was the pain made better by sitting forward, or did that make no difference at all?' Now, that may sound a total tautology, but it is aimed at preventing the patient from perceiving that you may have a bias for a particular reply – and, thinking you the expert, that is all too common.

Along the same lines, ask all of your questions from a positive rather than negative perspective. Phrasing a question such as: 'You didn't have any palpitations, did you?' strongly invites the patient to agree.

Also, don't ask two questions in one sentence; it only confuses the patient. E.g.: 'Did you have any chest pain or shortness of breath?'

Do not use medical terms, and if your patient uses them get him to explain what he means by them in terms of his symptoms. By the end of this stage you should have a good idea of *all* of the major symptoms the patient complains of, and the way in which they unfolded in time. This stage of *general* questioning needs no medical knowledge at all, nor should it be allowed to intrude.

Specific Interrogation:

The next phase aims first to determine more about the major symptom or symptoms involved — specifically the onset, severity, time-intensity relationships, precipitating factors for each episode, relieving factors, quality or character of the symptom, associated phenomena, and in the case of pain, its precise site and any radiation. In eliciting answers to these aspects, you should again ask only general questions, at least to begin with —e.g., it is far better to ask: 'Did you notice anything which made the pain worse?' than to ask: 'Was the chest pain made worse by exercise?' The latter immediately reveals your own bias, and is a loaded question. Only when your general questions do not elicit satisfactory answers, i.e. when all else fails, should your question be more specific. But even here, you must avoid loaded questions that show your own bias. . One way to do this is to give a list of alternatives, e.g. 'Was the pain throbbing, burning, cutting, stabbing, pressing, squeezing, gripping etc., in nature?'

Comment:

The reason for going into the principal symptom or symptoms in this way is to obtain an idea about two quite separate aspects of the condition facing you.

1. WHERE in the body, *anatomically,* is the condition *localised?*

At the very least we want to know if possible which *system* is involved, so that we can give that particular attention during our clinical examination. In this respect, the *site of* the symptom, its *radiation* (if pain), its *quality, precipitating, aggravating,* and *relieving factors,* are particularly important. Thus, a knife-like stabbing right lower anterior chest pain aggravated by inspiration and coughing, and relieved by holding the chest firmly, suggests an involvement, *anatomically,* of the pleura (or chest wall) overlying the middle lobe of the right lung. 'Crushing' central retrosternal chest pain radiating to the left arm, precipitated by exercise and relieved by rest, suggests disease of the *coronary arteries.* On the other hand, central chest pain worse in some positions and better in others, suggests that it may be stemming from a pericardial site. Yet other chest pain made worse on swallowing indicates an oesophageal origin.

2. **WHAT***, in genera l pathological terms,* is the *nature of the lesion?*

This is mainly given by the precise *mode of onset,* and the subsequent *time-intensity relationships* of the symptoms. This is discussed more fully below, but you should note that the onset is particularly important, i.e. whether hyper-acute, acute, sub-acute or chronic.

Systems enquiry:

This should not just be a mindless collection of a long checklist of all possible symptoms but should be approached from the perspective of clues already obtained. Particularly go into the other symptoms in the main system you suspect to be involved, e.g. cough, sputum, haemoptysis, shortness of breath, pain in the chest, weight loss, fever, in the respiratory system; shortness of breath on exertion, shortness of breath at night, orthopnoea, swelling of the ankles, chest pain, palpitations, faintness etc., in the cardiovascular system. It is true that you must ask about other systems, but only emphasise the positive and relevant negatives in your presentation.

Never forget in the systems enquiry to ask about *fever* and *weight loss,* because these are of great importance in making your general *pathological diagnosis.* Also ask about any change in secretions (e.g. yellow sputum suggesting local inflammation, etc).

Information from a third party:

This may be necessary in the young, or where the patient is a poor historian, or unconscious. (N.B. Never imply criticism of another doctor or hospital when discussing a patient's previous management on hearsay evidence from a third party. This is a very common cause of litigation and may get you into deep trouble.)

(2) Background History

Ask here about the factors that led up to the illness, because these may give you important clues to what predisposed to or precipitated it, relevant to the Aetiological diagnosis (see below). In doing so, again ask only general questions, at least initially. If specific questions do come into your mind (as they should), ask them, but only after the general ones, and again, do so in such a way as to not reveal that you have any particular answer in mind. There are a number of categories of questioning relevant to the underlying aetiological background of the disease, as follows:

Previous Illnesses:

Knowing about these can be very helpful. However, in discussing them, don't accept medical terms such as 'duodenal ulcer', 'angina', and 'gout'. Clarify all such statements from the patient by going into the symptoms precisely. When a febrile illness is involved, always ask about travel abroad. Ask also about previous chest X-rays and results of previous insurance examinations (e.g. blood pressure) where appropriate.

Family history:

Consider particularly that which might be relevant to the patient's present condition.

Social history:

Include occupation, alcohol, tobacco, home circumstances, interests, habits (including any dietary habits or fads), hobbies, etc. Think about the possibility of HIV infection, and tactfully ask questions correspondingly, in appropriate cases.

Psychological history:

It is best to broach this only towards the end of the enquiry when you have the patient's confidence. Indeed, obtaining a full history in the many cases where stress seems to be a feature may require coming back at a later stage. But finding out whether there are perceived stresses in the background is of the utmost importance, for these may not only bear on the reason for the precipitation of this particular disease, or disease bout, but it will almost certainly bear on the capacity of the patient to cope with it!

Drug history.

This is an increasingly important problem, both in terms of self-administration and medically prescribed drugs. If the patient is taking medications, get an accurate list; ask about any side effects, and the degree of compliance with therapy. Also, don't be afraid to ask questions, tactfully, of course, about use of illicit drugs.

The Beginnings of a Diagnostic Synthesis After History-Taking

At the completion of the history you should have gained a very good idea about which *system* is involved (i.e. the beginnings of an *anatomical diagnosis),* and a good idea about the *nature of the general pathology.* You should also have an idea of how the condition affects the patient because this is relevant to our third or 'Functional' category of diagnosis. You may very well have good information about the background aetiology as well, but remember that dissecting this adequately depends on your having a good idea of the anatomical and pathological nature of the lesion to begin with. This may not become absolutely clear until after the clinical examination, so you will often have to return to these background aspects of the history after completing your physical examination. Never forget that your first line of further 'investigation' should be a more complete history.

(1) Anatomical diagnosis

You should, by the end of the history, have a good idea of the system involved. Again, the quality, site, radiation, aggravating and relieving factors are most important in this respect. An example is that shortness of breath made worse by lying flat and relieved by sitting upright is generally of cardiac origin (left heart failure with pulmonary congestion). This example illustrates another aspect of making an anatomical diagnosis, namely that it is often functional consequences (i.e. the 'Functional' category of diagnosis) that give you the clue to

anatomy. Thus, shortness of breath does not point directly to cardiac involvement, but only indirectly through pulmonary congestion. So much of our anatomical diagnosis is dependent on knowledge of basic normal physiology. Again, the nerve dermatome distribution for pain, particularly referred pain, the physiology of the circulation with posture This is discussed more fully in **Part 1** of this document.

(2). General Pathological Diagnosis

(a) Hyper-acute onset conditions.

Those conditions that come on within a few seconds or minutes are called hyper-acute, and are usually brought about by rupture or obstruction of some hollow tube somewhere within the body. If the symptom goes away within a few minutes or hours, it is more likely to be obstruction than rupture. In some areas such as the abdomen there are many hollow tubes which can be obstructed or ruptured, including gut, blood vessels, gall bladder, ureter etc., but in others such as the brain there are very few, and sudden onset conditions there give the clue not just to pathology, but to the anatomical structures involved (see also Part 1).

(b) Acute conditions

These are ones that come on over a few days or up to about two weeks. If associated with fever, it is almost certainly an inflammatory condition (this is not to say it is necessarily infective, because any dead or damaged tissue, e.g. an area of infarction, may cause secondary inflammation).

(c) ***Sub-acute***, *say coming on over 2-6 weeks.*

(d) Chronic — i.e. coming on over months. In this case, the time-intensity relationships become very important. If relentlessly progressive, and particularly if associated with weight loss and no fever, *neoplasia* is likely. If associated with fever (with or without weight loss), then *chronic inflammation* is likely. In the latter case, look for confirmatory evidence of local inflammation in the system involved, both in the *secretions* (diarrhoea, purulent sputum, nasal discharge, vomiting etc.) or, if the region is accessible to touch and the patient's perception, pain, tenderness, heat, redness, swelling, loss of function etc., i.e. the hallmarks of *local inflammation.*Define the time-intensity relationships carefully, as this may tell you whether the disease is an acute remitting and relapsing process (such as so-called 'chronic' duodenal ulcer) or whether it is a chronic progressive one (like neoplasia).

(e) Long-term chronic conditions

When the symptoms have been present a very long time, neoplasia is unlikely, and the condition is more likely to be degenerative (e.g. emphysema), very chronic inflammatory (e.g. bronchiectasis), 'auto-immune', 'constitutional', hereditary, familial etc. These aspects of clinical pathology are also discussed fully in Part I.

Comment

Thus, even at the end of history-taking you should have at least some idea of what is going on in each of our four categories of diagnosis, particularly the first two. If you are lucky your diagnosis may almost be complete but, mostly, it will still be tentative at this stage. It should be written down in its four categories, as far as possible, e.g.

(i) Myocardial (anatomical), ischaemia (pathological).

(ii) With secondary angina, left heart failure, and atrial fibrillation *(functional* diagnoses).

(iii) Predisposed to by the *background* cardiovascular 'risk factors' of heavy cigarette consumption, high blood pressure, and diabetes, and with the possible precipitating factor of recent severe emotional stress.

Finally, having done this tentative diagnostic exercise, think about what further questions you should ask in the light of it, to help better define each of its diagnostic categories, before going on to the physical examination.

Writing up and presenting your history

In writing up and presenting your histories you should observe the following guidelines:

Presentation:

First present a *head-line,* including a brief statement to put the patient's background as a person in context and a succinct comment on his/her presenting complaint — e.g.: the patient is a 48 year old stressed housewife with six children who presents with a history of sudden onset of severe central chest pain radiating to the left arm, lasting for one hour, a week before admission, with five similar episodes since, all coming on out without obvious reason.

Present illness*:*

This should *begin at the beginning* and give a chronological account of the problem(s). By all means present a brief background at this stage if you wish, but get to the *major symptom* early on and elucidate the way it unfolded in time (time-intensity relationships), followed by the other aspects of that particular symptom as already discussed, i.e. its site, radiation, quality, severity, and precipitating, aggravating, and relieving factors. Then obtain the history about *other symptoms related to the system that* seems to be involved (e.g. in the case of cough: shortness of breath (including positional variation), sputum, haemoptysis, chest pain, weight loss, fever etc.). In this respect, it is just as important that you comment on the absence of potentially associated symptoms as their presence.Only after this should you go into *enquiry about other systems.* Most students spend far too much time on the latter and far too little on the main problem, so that the history loses its perspective.

At each stage of the presentation you should pause and give yourself an opportunity to elucidate any important points that have come up because of the information already obtained (e.g. if you think the patient has pericardial pain for some reason, then it is important to ask about the presence or absence of aggravation of the chest pain by posture).

Background history*:* as above.

Example of a written/oral history presentation

The following is a sketch of a possible history, put forward to amplify the above. It is not meant to be complete, but to indicate the *framework* you should use.

Presentation:

The patient is a 46 year old housewife with six children, who smokes 60 cigarettes per day, and presents with a history of five episodes of severe central chest pain in the past week.

Present illness:

The patient had been well until a week before admission. At that time, on a Saturday evening, and after having had a rather harrowing evening at home with 'friends', she noticed the sudden onset, over a few minutes, of an increasingly severe 'heavy' central chest pain, spreading over the next few minutes to involve the left shoulder and the upper aspect of the left arm, where it felt not only 'heavy' but 'numb'. That particular episode passed within five minutes, and she gave it no further thought until two days later, when it recurred, this time whilst she was hanging out her washing on the Monday morning. This episode was more severe than the last, and caused her to sit down, after which it slowly eased over the next 15 minutes. Over the next five days there were several more episodes, each about the same as the others in site and radiation, but varying from 10 minutes to half an hour in duration. She noticed no precipitating factors (in particular, there was no relation to effort), and no factors that aggravated the pain once it had commenced. She could do nothing specific that would bring relief. In between attacks, she felt perfectly well.

There were no associated symptoms, either during or between the attacks, except for rapid palpitation and shortness of breath soon after the start of one attack. There was no other shortness of breath, either on exertion or at night, no discomfort on lying flat. No palpitations, no faints, no swelling of the ankles, and no blackouts.

Systems enquiry revealed very little. The result of specific interrogation were as follows:

Cardiovascular system:

As above: in addition, no claudication; no history of varicose veins.

General:

No tiredness, loss of energy, weight loss, loss of appetite, or change in sleep pattern. No shivers or sweats.

Respiratory:

No cough, sputum, haemoptysis, chest pain, shortness of breath, or wheeze.

Alimentary system:

No loss of weight or appetite, no difficulty in swallowing, no indigestion, no abdominal pain or vomiting, no disturbance of bowel habit, no P.R. bleeding or jaundice.

Urinary system:

No increased frequency of micturition, no nocturia, no dysuria, no haematuria.

Genital system:

Periods normal. Six previous pregnancies — all full term and normal children of normal weight. The patient had been taking oral contraceptive therapy for 7 years.

Haemopoietic system:

No anaemia, spontaneous bruising or bleeding episodes.

Endocrine system:

No thirst, polyuria, pigmentation, goitre, weakness.

Skin:

No rashes, pustules or boils.

Locomotor system:

No joint pain or swelling. No weakness.

Nervous system:

No headaches, 'turns', fits, visual speech or hearing disturbances, no weakness, imbalance, or sensory symptoms; normal sphincter control. No recent change in behaviour or mood.

Past history: Past history of high blood pressure, during all pregnancies. Blood pressure had not been taken since the last pregnancy 7 years ago. No history of diabetes.

Family history: Mother died 56 years of a 'stroke' (had high blood pressure). Father died aged 61, of 'heart attack' (also hypertensive). One sister (younger) on treatment for high blood pressure

Social History: Husband an alcoholic; many family difficulties. Patient complained of a great deal of anxiety and worry, particularly over the welfare and future of her children. Normally smokes 20-25 cigarettes per day, but up to sixty in recent weeks, after domestic problems increased. She had recently started drinking up to 'two or three glasses' of cask wine per day.

Psychological history: As above.

Drug therapy: a high dose oestrogen oral contraceptive therapy for 7 years.

Comment:

Thoughts on diagnosis at this stage could be jotted down as follows:

1. *Anatomical* diagnosis: The site, radiation, quality and nature of the pain would suggest a myocardial origin.

2. *Pathologically,* this is an acute relapsing and remitting process (i.e. reversible *ischaemia*).

3. *Functionally,* there seem to be very few consequences, except for possible secondary dysrhythmia and left heart dysfunction in one attack.

4. The long-term background upon which these symptoms have arisen (and perhaps relevant to underlying *aetiology)* are a past history and family history of hypertension, heavy cigarette smoking, oral contraceptive therapy. And the unusually high amount of psychological stress recently may need to be considered as a possible precipitating factor for the recent acute illness. (It will certainly need to be dealt with in its own right, for the

patient's future ease (i.e. lack of 'dis-ease').

Of course, no diagnosis could be complete at this stage, and must await more information from the clinical examination. Even then, more history may be needed for a full clinical diagnosis (especially in the Aetiological category).

N.B. When any patient seems to have two or more clearly different and unrelated problems, it is sometimes better to obtain and present the history related to each quite separately, and discuss the diagnostic aspects of each within the framework of a Problem list.(But always be on guard that you may even then be dealing with multiple manifestations of single organ or system disease).

CONCLUSION

Show that your history has found the wood through the trees. Avoid medical jargon in presenting it. Be clear and concise both in writing up and presenting your histories. Always avoid bias, not only in your questioning, but also in interpreting the symptoms. Use the clinical information *in each individual patient* to draw conclusions, and never jump to conclusions on the basis of small pieces of evidence and then try to justify them by some theoretical knowledge you may have. Knowledge of physiology is to be used in *interpreting* the *individual patient's* symptoms, not in making the patient fit your preconceptions! (See also the 'Making Physiology Work in Clinical Diagnosis online computerised tutorial system at this same website address, viz:

http://www.healthsci.utas.edu.au/medicinenew/staff/Boyd/gbhp.html).

Observe *courtesies* to patients and staff. Don't discuss problem medical areas in front of patients. Terms like 'cancer', 'Ca'. 'malignancy', etc. are best avoided ('neoplasia' is best). Get their confidence, make them feel relaxed; be helpful and unhurried, and always remember Ericson's truism:

> "The best kind of history-taking is at the same time the best kind of psychotherapy."

Part III Physical Examination of the Various Bodily Systems

Introduction:

The purpose of this section is to give you a framework to approach physical examination so that you may collect all the relevant information, and do so *accurately, thoroughly* and completely

Category A.

The recommended ***order*** of performance ***and techniques of physical examination*** you should master within each of the various systems
(left hand column).

.Category B

Physical signs

(right hand column).

The technique and order of performance of physical examination of the various systems.

This will be shown on the left-hand page of each section, and is most important. It relates to the techniques that you should master, and the *order* in which I recommend they be performed within each system.

These are the ordinary basic *routines* of physical examination. By the end of your introductory clinical training, you must be able to carry out all of these techniques *competently, without hesitation,* and *without any omission.* Of course, in subsequent years, we expect you to be able to perform more than just the *routine*, such that, like having leant the routine of driving a car, your senses can automatically become free to observe and interpret other relevant information along the way. Even that can present a problem in a patient with a lot of physical findings, so remember to pause at the end of each phase of the physical examination of each system (e.g. after examining the hands routinely, and ask what more, in the light of information already gathered, you should be looking for).

It has to be said that the layout within Category A tends to be somewhat artificial, in that we usually start by teaching examination of different systems separately. Eventually you will have to learn to do a *complete* physical examination, and because of that, the broad routine of this process, which covers all of the systems, is outlined at the end of this section.

Physical signs which you should be able to elicit and interpret. .

Experience has shown that students approaching their final examination in Medicine often have a very good knowledge about medical conditions, yet be far from competent in carrying out a physical examination and using the data so obtained to diagnose various clinical conditions. One of the problems in this respect is that students are not always aware of just how far they fall short of the expected standard in relation to clinical signs, and this category aims to improve this by stating the minimum range of clinical signs to be recognised and understood. Students should be able to diagnose all of the *common* conditions from physical signs by the end of their course. This section should be used as a checklist for this purpose. Every effort should be made to fill in any gaps, and the aid of your tutors should be sought in this respect. Photographic slides, audio-visual, computerised tutorials and other aids can also help, especially with less commonly observed, yet potentially reversible and therefore important conditions. But in the end there is no substitute for seeing patients on the wards - including on a self-directed basis. It is, in the end, up to you

Cardiovascular System

1. GENERAL APPEARANCE, including face, tongue, mucous membranes. From the end of the bed, with clothes removed down to underpants/briefs. As with all subsequent steps, first look with the open mind, then close your mind and look for specific signs in the light of clues already gleaned from the history etc	1. GENERAL APPEARANCE Colour, including cyanosis, anaemia etc. Dyspnoea/ hyperventilation. Pulsations, particularly in neck, precordiurn and epigastrium. State of ease or otherwise. Malar flush of mitral stenosis. Skin cool, atrophic, with hair loss (espec. lower limbs) in peripheral vascular disease
2. HANDS • examine carefully	2. HANDS Cyanosis, central versus peripheral. Anaemia (palmar creases). State of perfusion of periphery (hand warmth). Clubbing of nails. Splinter haemorrhages. Osler's nodes.
3. PULSE (Radial) Rate, rhythm, regularity, pulse volume, wave-form	3. PULSE Arrhythmias, including sinus arrhythmia, atrial fibrillation, extra-systoles, coupled beats (pulsus bigeminus). Also pulsus paradoxus (in severe asthma, constrictive pericarditis). Pulsus alternans (severe hypertension). Collapsing pulse (at wrist) in aotric valve regurgitation. Slow-rising pulse (best felt at neck) in artoic valve stenosis. Bisferiens pulse of aortic stenosis with combined incompetence. Tortuous visible arteries (e.g. locomotor brachialis). Osler's sign (palpable radial artery wall) in pseudo-hypertension.
4. BLOOD PRESSURE (Sitting and standing, with pulse in each position).	4. BLOOD PRESSURE Recognise response to standing (slight fall in systolic, slight rise in diastolic; increased pulse rate) i.e. baroreceptor reflex.
5. JUGULAR VENOUS PRESSURE (a) If normal: demonstrate that it is venous by gently laying your index finger across the base of the neck and seeing the vein distend from above; observe how the pulsation at the base of the neck obliterates with slight pressure. Look for the double impulse and the variation in height with inspiration, upright posture, and hepato-jugular reflux. (b) If JVP elevated: go on to feel for pulsation in the liver, particularly if prominent V wave. Also look for spleen, ascites and oedema. (c) If in doubt, sit the patient bolt upright, when the top of a very elevated venous pulse may become visible for the first time.	5. JUGULAR VENOUS PRESSURE Diagnose and measure height of any raised JVP. Note any exaggeration of 'a' or 'v' waves; time and interpret these (absent 'a' wave in atrial fibrillation; large 'v' wave in tricuspid valve regurgitation; intermittent 'cannon' waves in complete heart block. Signs of obstruction of the superior vena cava. Kussmaul's sign in constrictive pericarditis, right ventricular infarction.

6. CAROTIDS Feel with thumb, particularly noting upstroke velocity. Listen for carotid murmurs.	6. CAROTIDS. Slow upstroke of carotid pulse in aortic valve stenosis. Low-pitched bruit radiating from base of heart in aortic stenosis. High-pitched long loud localised bruit (upper border of thyroid cartilage) in internal carotid artery stenosis. Distinguish arterial bruits from venous hum (the latter will disappear on gentle pressure over the jugular veins).
7. MEDIASTINUM Define if trachea is mid-line (determine by pressing middle finger straight back but angled downwards, just above the sternal notch).	7. MEDIASTINUM Deviated to the side of any collapsed lung. Deviated away from the side of a large pleural effusion.
8. HEART a). Inspection for visible apex beat, and for any abnormal pulsation.	8. HEART (a) Inspection Visible pulsations
b). Palpation - not just of the apex, but pulsation elsewhere. Also thrills (palpable murmurs, like a cat's purr). N.B. If in doubt, here or in any other category a) - d) (especially auscultation), ask the patient to stop breathing for a few moments.	(b) Palpation Apex beat. Characteristic thrust at apex in LV hypertrophy; left parasternal heave in RV hypertrophy. Abnormal pulsations elsewhere (e.g. cardiac aneurysm, left ventricular dyskinesia after myocarduial infarction). Thrills, including where maximal, and timing
c). Percussion. Percuss especially where apex beat impalpable, and where pericardial fluid suspected.	(c) Percussion. Practice your technique.

d). Auscultation - room must be quiet! - First listen to 1st and 2nd heart sounds with bell at apex (pressing only very lightly). Next listen to 2nd sound at base with diaphragm, and define its split with inspiration. Now listen for added heart sounds at the cardiac apex and left parasternal area, especially with bell for 3rd heart sounds, 4th sounds. Then listen for murmurs (ask the patient to stop breathing, in mid expiration, if you are having difficulty), first over the whole precordium (including left infraclavicular region) with both bell and diaphragm in a general way. Then listen specifically for: Apical murmurs. Systolic ones with the diaphragm. Basal murmurs. Most heard best with diaphragm. Also listen in neck. Diastolic. Listen for murmur of mitral stenosis (with bell), especially if a loud first heart sound or an apical pansystolic murmur is heard. Also listen (with diaphragm para-stemally) for aortic valve incompetence Remember with all murmurs to describe its site, where maximum, its radiation, quality, pitch, intensity, position and duration in the cardiac cycle, and what if any manoeuvres alter its intensity. Make two postural manoeuvres on routine auscultation. First, place the patient on left side and listen at the apex for mitral stenosis (with bell); then with the patient sitting up and breathing out, listen at the aortic area and down the left parasternal edge for the early diastolic (high-pitched blowing) murmur of aortic valve incompetence. This latter should be the last part of the cardiac examination, for the patient is now sitting up and you can then conveniently go on to the next stages (9 and 10). Know how to perform valsalva manoeuvre, handgrip, and be aware of other manoeuvres which may help in diagnosis by accentuating or diminishing some murmurs.	d) Auscultation. Gently with the bell; more firmly with the diaphragm. Heart sounds. Not usual to be able to detect split first sound. Recognise split second sound, increased split during inspiration. Third heart sound in normal children, and with cardiac dilatation in adults (volume overload). Fourth heart sound in ventricular hypertrophy, or other cause of stiff left ventricular wall. Murmurs: Be able to diagnose classic murmurs of mitral stenosis, mitral incompetence, aortic stenosis, aortic incompetence, VSD, tricuspid incompetence; (tricuspid stenosis, pulmonary stenosis, pulmonary incompetence, patent ductus, ASD). Also friction rubs (pericardia!, pleuro-pericardial). Pansystolic murmurs include mitral incompetence, tricuspid incompetence (increased by inspiration), VSD. Ejection systolic murmurs include aortic and pulmonary valve stenosis. Diastolic murmurs Include mid-diastolic at apex in mitral stenosis, and early diastolic at left strnal edge in AI (also, rarely, pulmonary incompetence). Be able to comment on the haemodynamic significance of all valve lesions. Use of Valsalva, handgrip, respiration, and other manoeuvres to bring out particular murmurs (see also ‘Making Physiology Work in Clinical Diagnosis’ online at this same website address.
9. LUNG BASES Percuss bases and listen for breath sounds and inspiratory crepitations (crackles). When describing breath sounds always describe all of the following: their quality, intensity and the presence or absence of any added sounds. If crepitations present, define their position in respiratory cycle, and see whether coughing can clear them.	LUNG BASES Detect fine, late inspiratory crepitations (crackles) of early pulmonary oedema, and distinguish from ‘normal’ (the latter sometimes present in immobile or elderly patients, but are cleared by coughing or a few deep breaths - ‘physiological’ creps.).
10. SACRAL OEDEMA Look for this especially in patients confined to bed.	10. SACRAL OEDEMA Pitting versus non-pitting oedema. Unilateral oedema and calf tenderness in DVT
11. THE VASCULAR SYSTEM Listen for an abdominal bruit in hypertension and feel for radio-femoral delay. Palpate the abdominal aorta. Examine femoral, popliteal, and foot pulses, (dorsalis pedis and posterior tibials). Look for ankle oedema; calf tenderness. Examine state of perfusion of legs, including Buerger’s test where indicated	11. THE VASCULAR SYSTEM State of veins (including varicose veins). Skin temperature, atrophy, nail and hair growth (reduced in peripheral vascular disease). Calf tenderness in deep venous thrombosis

12. NECK Listen in the neck for bruits, especially over internal carotid artery origin, if you have not already done so	12. NECK bruits Distinguish carotid artery stenosis, from aortic valve stenosis (with radiation of the murmur to the neck).
13. OPTIC FUNDI Examine the retinal vasculature, noting particularly arterial lumen calibre and regularity, light reflex from artiries; a-v nipping; haemorrhages, exudates, papilloedema.	13. OPTIC FUNDI Note A/V ratio, particularly arterial narrowing (look along the artery to see whether this is uniform or irregular). Look for increased light reflex from arteries, e.g. copper wiring, silver wiring. Haemorrhages of different types (blot, dot, flame-shaped, sub-hyaloid) and their origins. 'Hard, and 'soft' (retinal infarcts) exudates. Optic atrophy. Papilloedema
COMMENT This should be the order of a complete routine physical cardiovascular examination. However, at the end of each step you should pause to ask what in addition you should look for in the light of the history and/or other clues in this particular patient, e.g. listen particularly for a pericardial friction rub in any patient with chest pain aggravated by lying flat and eased by sitting forward. As each stage of the routine examination is completed, ask yourself whether there are particular things you should be looking for in this way. Also, at the end of the examination, are there now more questions you wish to ask in the history related to any of the four categories of diagnosis, viz. Anatomical, Pathological, Functional, Aetiological, particularly the latter?	COMMENT Be able to diagnose: Congestive cardiac failure (high and low output); left and right. Ventricular hypertrophy/failure Cardiac valvular lesions/shunts. Hypertension Myocardial infarction Sub-acute bacterial endocarditis Hypotensive shock - with and without peripheral vaso-constriction Vena caval obstruction, superior and inferior Constrictive pericarditis Cardiac tamponade

Respiratory System

1. GENERAL EXAMINATION From the end of the bed (with clothes removed at least above the waist). First open-mindedly looking for any abnormality at all; then with a more closed mind looking for cyanosis, respiratory distress, use of accessory respiratory muscles, asymmetrical chest movements etc. Sometimes easier to perform chest examination with patient seated on a stool. Hands on head also helps examine axillary areas	1. GENERAL EXAMINATION Respiratory distress. Use of accessory muscles of respiration. Wheeze (sometimes audible in asthma). Inspiratory stridor in tracheal obstruction Cyanosis. Asymmetry of chest movement.
2. FACE/TONGUE	2. FACE/TONGUE Cyanosis - central vs. peripheral (in the physiological sense).
3. NASAL SINUSES	3. NASAL SINUSES Detect fluid in maxillary sinuses.
4. VOICE	4. VOICE Husky voice in recurrent laryngeaJ nerve paralysis. Nasal voice of palatal paralysis.
5. COUGH	5. COUGH Moist or dry. Brassy' or 'bovine', weak cough in recurrent laryngeal nerve paralysis
6. SPUTUM Examine	6. SPUTUM Mucoid, mucopurulent, purulent, blood-stained.
7. HANDS Particularly look for finger clubbing, cyanosis, state of skin perfusion. Any tremor, especially of hyperextended hands.).	7. HANDS Central vs. peripheral cyanosis. Clubbing. Signs of CO2 narcosis (metabolic flap/asterixis etc.).
8. TRACHEA Position and length of trachea above sternal notch	8. TRACHEA Normally very slightly deviated to right. Very little trachea above sternal notch in chronic obstructive airways disease.
9. LYMPH NODES Axilla from front, neck from back.	9. LYMPH NODES Axillary, supraclavicular, and infraclavicular. Define any enlargement; also consistency, matting of nodes, size, number distribution etc.

10. CHEST Inspection. Degree and symmetry of movement, type of respiration (patient at 45 degrees); degree of resting inflation (patient sitting upright). Upper chest movement best observed by kneeling at the end of the bed whilst the patient lies flat.	10. CHEST (a) Inspection Shape of chest - barrel chest, kyphoscoliosis. Recognise changes in respiration: Increased rate and/or depth Use of accessory muscles of respiration Indrawing of intercostal spaces in severe airways obstruction Prolonged expiratory phase in airways obstruction Grunting respiration of pneumonia with pleurisy. Kussmaul respiration. Cheyne-Stokes respiration. Stridor of trachea! stenosis or obstruction. Other: Asymmetry of chest movement Recognise lobar surface markings Abnormal veins on chest wall, colour transition in SVC obstruction.
(b) Palpation. Chest movement (including symmetry). Remember to palpate anteriorly in three positions, placing hands symmetrically: (i) Under the clavicles (ii) In the mid-chest anteriorly, over the breasts, and (iii) Laterally, under the breasts. Measure chest expansion with tape measure at the normal nipple line. Vocal fremitus	Palpation. Alterations in movement, symmetry. Vocal fremitus. Rib lumps; rib tenderness, local or on compression (eg. with fractures).
(c) Percussion — use clavicles as well. Compare each side as you go along. Perform with ‘follow-through’ action. Percuss para-stemally, then laterally, including axillae. Light and heavy percussion. Liver and cardiac dullness. Remember to percuss apices (above clavicles).	Percussion. Degree of dullness. Tidal percussion: movement of percussion borders on inspiration, (e.g. liver dullness).

(d) Auscultation - most use stethoscope diaphragm, but use bell in hairy patients. - Again remember to give a description of all three major aspects, viz., the intensity of the breath sounds, their quality, and the presence of added sounds (crackles, wheezes etc). If added sounds are present, determine type, effect of coughing, and position in respiratory cycle. Remember apices. Vocal resonance	Auscultation (i) Breath sounds. Normal breath sounds are vesicular, and produced by airways rather than in alveoli; Intensity related to total and regional airflow. Reduced breath sounds occur with obstruction of regional airflow (e.g. bronchial occlusion) or attenuation at an interface (e.g. pleural effusion), emphysema. Increased breath sounds: (bronchial breathing, aegophony, whispering pectoriloquy), occur with reduction of normal regional attenuation of breath sounds (e.g. in lung consolidation, dense pulmonary fibrosis). (ii) Vocal sounds. Vocal resonance. Produced and altered as above. (iii). Added sounds. Note type, pitch, intensity and position in respiratory cycle. Crackles. Coarse; in large airway secretions, inspiratory and expiratory, cleared by coughing (thus distinguished from pleural rub). Fine creps/crackles. are produced by sudden opening of peripheral airway units. Late high-pitched basal inspiratory creps suggest alveolar involvement (e.g. pulmonary oedema). Early inspiratory and late expiratory creps suggest origin from small to medium airways, e.g. in bronchitis/bronchiolitis. Wheezes. Produced by oscillation of opposing airway walls with significant narrowing. Pitch determined by physical properties of airways rather than airway size Monophonic wheeze in local obstruction; polyphonic in widespread obstruction. Wheezes usually expiratory: inspiratory wheeze implies significant airway narrowing (as does pulsus paradoxus - therefore check for this where appropriate, e.g. severe asthma). Pleural rub - distinguish from coarse crackles, (ask patient to cough).
11. GENERAL COMMENTS Often helpful to examine chest with patient straddling a chair/stool. Remember to examine the lateral aspects of chest, especially in axillae (hands-on-head helps).	11. GENERAL COMMENTS Know the surface markings of the major lung lobes. Know accurately the physical signs of pulmonary collapse (with and without an obstructed bronchus), pulmonary consolidation, pleural effusion, pneumonthorax, and emphysema. Also pulmonary fibrosis, pulmonary embolism, and cor pulmonale. Be aware of normal increase in breath sounds at right apex (due to trachea being slightly to right).

Alimentary System

ALIMENTARY SYSTEM EXAMINATION	ALIMENTARY SYSTEM SIGNS
GENERAL - make sure all clothes are removed, down to underpants only. 1. GENERAL INSPECTION (from end of bed) Hands, skin, face, abdomen.	1. General Inspection. State of nutrition, hydration, any tremor or restlessness, jaundice, pigmentation, hair loss, spider naevi, bruising, gynaecomastia, parotid enlargement.
2. HANDS Including palms, nails, colour.	2. HANDS Inspect palmar creases as an index of anaemia (and confirm by looking at conjunctivae, tongue, areolae). Koilonychia in chronic iron deficiency. Leuconychia, finger-clubbing, palmar erythema, bruising, Dupuytren's contracture in chronic liver disease. 'Metabolic flap' in hepatic failure (also seen with CO_2 narcosis, renal failure).
3. MOUTH Including throat, tongue, teeth, and gums.	3. MOUTH Pigmentation of the buccal mucosa in Addison's disease of the adrenal glands. Pigmentation of the lips in Peutz-Jegher's syndrome. Koplik's spots in measles. Petechiae over the junction of hard and soft palate in infectious mononucleosis. Dry tongue in dehydration (also mouth breathing). Glossitis in nutritional deficiencies, pale tongue in anaemia. Atrophic tongue also in most anaemias (not just B 12 deficiency). Large tongue in amyloid disease, acromegaly.
4. SALIVARY GLANDS Parotids, submaxillary glands especially. Also inspect and palpate parotid duct orifice.	4. SALIVARY GLANDS Enlargement from lymphocytic infiltration in lymphoma etc. Enlargement in Sjogren's syndrome (also dry eyes). Parotid enlargement in alcoholic liver disease
5. ABDOMEN Lie patient flat, remove blankets, and turn down sheet to level of symphysis pubis. Inspection - respiration, symmetry, contour, swellings, distension, etc. Looking tangentially across abdomen during quiet respiration often helps define subtle masses even at this stage.	5. ABDOMEN (a). Inspection. Observe any alteration in contour, particularly fullness in the flanks (fluid?), other masses, and asymmetry. Look for any visible scars, marks, striae, visible peristalsis or pulsation; abnormal veins including caput medusa (veins spreading from the umbilicus in portal hypertension). Also jaundice, loss of hair and female distribution of bodily hair in chronic liver disease/alcoholism.

Palpation. Ask patient if they have any tenderness; then palpate in each quadrant (warm hands), first lightly then more firmly. Keep observing the patient's eyes for any signs that you may be hurting. Start in left iliac fossa, and then work anti-clockwise. Perform with your interphalangeal joints extended, flexing at M-P Joints only. Note masses, firmness, guarding, tenderness, and pulsation. It often helps if you sit on a chair or kneel with one knee on floor when palpating abdomen. *Comments:* .If any *mass* felt my palpation, then determine its *anatomical localisation,* namely whether it is attached to the diaphragm and whether it is an anterior or posterior organ. Attachment to the diaphragm given by movement (and direction of movement) on inspiration. Anterior vs. posterior organ given by where dull to percussion (anteriorly or posteriorly), Whether you are able to ballott the mass from the loin (if so probably posterior, but large anterior masses sometimes also ballottable). If you can only feel a vague mass on initial examination *always* go on to do a bi-manual examination, including ballottment. This especially helps with posterior masses. Routine palpation of organs. Feel specifically for liver, spleen, kidneys, (aorta, gall bladder). Bi-manual helps define liver edge better. Getting the patient relaxed, both your hands in position (keep them still) and succeeding to get the patient to take deep breaths with an open mouth are vital to tipping a spleen. Also, don't palpate too laterally for a spleen. It may be felt more medially than you expect, especially if moderately large. Right lateral position may help in feeling a difficult spleen. Bi-manual examination with ballottment at the very end of inspiration is the way to feel for enlarged (or displaced) kidneys, (can also help greatly in detecting splenic enlargement).	Palpation. Observe any tenderness, guarding or release tenderness. Observe any masses and describe their anatomical localisation and physical characteristics, including contour, regularity, smoothness, firmness, tenderness, etc. (see also Category A). Then look specifically for the liver edge (if felt, is it displaced or enlarged, i.e. percuss from upper to lower border -normal percussion span about 12 cms). Look for spleen enlargement (particularly in lymphoproliferatlve and myelo-proliferative disorders, portal hypertension, severe CCF). Enlarged kidneys (especially in polycystic disease). Palpable pulsatile aorta (also listen for overlying bruit). Gall bladder - if enlarged, is sometimes easier to see than feel, especially by looking tangentially across the abdomen when the patient takes a deep breath (alternatively try standing at the patient's right shoulder and feeling with the cupped right hand over his/her right hypochondrium during inspiration). Remember Courvoisier's law - if gall bladder enlarged; also Murphy's sign. Recognise palpable sigmoid colon, caecum, with constipation. Gastric sucussion splash in upper intestinal obstruction. Remember the five 'fs' in any abdominal distension (fat, fluid, flatus, faeces, foetus).
(c) Percussion. Routine, and over all palpable organs. Remember to percuss upper and lower limits of liver dullness; also percuss bladder where appropriate. Demonstrate resonance In flanks in normals; shifting dullness if fluid suspected.	Percussion: Liver dullness, splenic dullness (normally no dullness further anteriorly than the left mid-axillary line). Shifting dullness in ascites; fluid thrill in tense ascites (where you may have to use a bi-manual 'dipping' technique to feel any enlarged organs).
(d) Auscultation — over any mass; bowel sounds, bruits	(d) Auscultation — increased bowel sounds in gut obstruction, absent in ileus. Vascular bruits including aortic, femoral, renal and other arterial obstructions. Hepatic bruits (over vascular secondaries, hepatoma). Splenic friction rubs.
6. HERNIAL ORIFICES, INGUINAL AND FEMORAL LYMPH NODES	6. HERNIAL ORIFICES, INGUINAL AND FEMORAL LYMPH NODES Always inspect hernial orifices and feel for enlarged *inguinal and femoral* chain lymph nodes separately, describing their consistency, size, degree of matting etc.
7. GEN1TALIA Always inspect as part of the routine examination.	7. GENITALIA Testicular atrophy in chronic liver disease, etc.

8. RECTAL EXAMINATION Including stool inspection and test for occult blood. Remember: 'If you don't put your finger in it, you'll put your foot in it!'	8. RECTAL EXAMINATION Rectal masses, enlarged prostate (benign versus malignant). Haemorrhoids, tenderness (e.g. pelvic abscess or other inflammation). Recognise stool in malabsorption, melaena, iron therapy, obstructive jaundice, and ulcerative colitis.
9. GENERAL COMMENT Always give a statement about the (i). Anatomical localisation of the organ, before naming it, e.g. this mass moves downwards with respiration, is resonant to percussion anteriorly and ballotts from the loin; therefore it is a posterior mass attached to the diaphragm, and most likely kidney. Do not do the reverse of jumping to a conclusion and trying to justify that by subsequent examination. If you do, one day you will find a mass which does not fit your preconceptions and be confused, e.g. a central abdominal mass which is mobile, does not move with respiration but is dull to percussion anteriorly is, using your anatomical knowledge, probably a mesenteric mass (? small bowel mesentry, omentum, transverse colon); however the pattem-recogniser will find diagnosing this difficult, because he hasn't seen its like before. (ii). Pathology - apart from major information on anatomy, you can also often get some indication of the pathology involved by the character of the mass, i.e. whether cystic or solid, irregular, hard, craggy, ulcerated etc. In this respect you should also look at secretions, in this case the faeces; e.g. blood suggests ulceration; the presence of neutrophils microscopically provides important evidence of (bacterial) infection. Always look in any system for both the *general* (fever) + *local* (heat, redness, swelling, pain and tenderness, loss of function, purulent secretions) evidence of inflammation - in the abdomen the latter includes rigidity, guarding, tenderness, release tenderness, and/or (purulent) diarrhoea.	9. GENERAL COMMENT Be familiar with the external and general manifestations of failure of the major intra-abdominal systems including chronic hepatic failure, chronic renal failure, and chronic malabsorption. Also recognise conditions that may cause infiltration, particularly of liver, spleen, and lymph nodes, without overt clinical signs of chronic failure of the organ concerned. Recognise and be able to differentiate firm, smooth enlargement of an infiltrated liver from the moderate irregular enlargement of cirrhosis, and this in turn from the large craggy hard nodular and very irregular enlargement of secondary carcinoma of the liver. Know the signs of hepatic failure, ulcerative colitis (including acute toxic megacolon) and of obstructive jaundice (remember to examine urine as well as stool).

Nervous System

GENERAL First inspect and palpate the area indicated by the history to be Involved, to decide which system to examine in more detail. For example, in a patient with leg weakness, do not assume that you are dealing with a central nervous system problem. Inspection and palpation might reveal muscle tenderness and wasting more compatible with a primary *muscular* inflammatory disorder, for example (if there is doubt, you should ask about sensory disturbances, which would clinch a central nervous system rather than a primary muscular cause). Alternatively, there may be serious joint disease with secondary wasting and weakness of muscles, so be aware of this. Another important principle is to examine the affected part both at rest and under load, because this may bring out the symptom, e.g. weakness of the legs, arms, or ocular muscles under load in myasthenia gravis. This principle of examination under load is one you should always bear in mind with the examination of any system (for example exertion may bring out obvious signs of heart failure not present at rest in borderline cases).	GENERAL Inspection, including state of consciousness. Also inspect face and limbs, looking in the latter particularly for muscle bulk, fasciculation, tremor and other involuntary movements, spasms, tics, convulsions. General inspection should include skin, hair distribution, and pigmentation. Also skull inspection, palpation, percussion; auscultation (especially over orbits, carotid bifurcations). Inspection under load, e.g. walking (gait, ataxia, weakness, foot drop etc.; fatigue under load in myasthenia gravis, etc.).
1. HIGHER FUNCTIONS (a) State of consciousness and mood, including comprehension, insight and self-awareness.	1. HIGHER FUNCTIONS (a) State of consciousness and mood Abnormal levels of consciousness – alert, drowsy, stupor, coma. Alternatively, note increased activity, e.g. in hyperthyroidism, hypomania, anxiety; agitation of alcohol withdrawal in delirium tremens. Altered mood includes emotional change, confusion, illusions, hallucinations, delusions, and flights of ideas, depression, euphoria, anxiety, indifference, and catatonia. Importance and interpretation of Doll's eye reflexes in normals and various coma states

(b) Higher cerebral function, including mini-mental state examination. *Orientation* in time, place and person. Name the year, season, date, day, and month. Where are we: State, city, suburb, hospital, and floor/ward? *Registration*– Name 3 objects; 1 second to say each. Then ask patient to repeat all 3. Or repeat until all 3 learned. *Attention and calculation; s*erial 7s subtracted from 100; alternatively get patient to spell 'world' backwards, or do monetary calculations. Repeat 7 digits forwards; 5 digits backwards. *Recall.* Ask for the 3 objects in (ii) above to be repeated. Similarly, short-term memory can be tested by the Babcock sentence. Alternatively, the patient should normally be able to repeat any sentence that includes a name, address, and an object 5 minutes after 1st learning. *Short-term memory*. E.g. name contemporary events; popular figures. *Language skills*. Eg. Name a pencil and a watch. Repeat: 'No ifs, ands or buts'. Follow a 3-stage command. Read and obey a simple command Copy a sentence Copy a design	(b). Higher cerebral function, including mini-mental state examination. Be aware of the patient's background before setting standards. As with other aspects of intellectual function, if in doubt enquire from relatives about any deterioration they have noticed. <u>Scoring the result of mini-mental state examination</u> *Orientation.* Score 1 point for each correct answer out of 10 items asked in this category. *Registration.* Score 1 point for each correct answer to the 3 items asked in this category. (*Attention/calculation.*Score a max. of 5 points for correct serial 7s, or 'world' spelled backwards. *Recall*. Up to 3 points for correct recall of 3.objects. *Language.*- 2 points for naming 2 objects. 1 point for repeating 'No ifs, ands or buts' Up to 3 points for following correctly a 3-stage command. Up to 3 points for correcting reading and obeying the 3 commands Maximum score = 30 marks. Make sure the patient is fully alert, ie. not confused or disorientated because of some temporary condition before branding him/her with any label of cognitive impairment/ dementia.
(vi). *Abstract reasoning.* Ask meaning of proverbs; get patient to explain difference between, say, a child and a dwarf; or the difference between a lie and a mistake; other analogies. . (vii) *Language and articulation* <u>Dysphasia</u>. Determine whether left or right handed; ability to understand spoken words; ability to express in speech; ability to understand written words or commands; ability to express in writing. Distinguish dysphasia from dysarthria.	(vi). *Abstract reasoning.* Proverbs are useful, but many people know their meaning from long ago, and other analyses of concepts, particularly use of analogy, can be helpful e.g. 'Why can the heart likened to a pump. (vii). *Language and articulation* Recognise the difference between <u>receptive</u> or fluent aphasia (often containing jargon which the patient does not have insight into) in receptive dysphasia (Wernickie's area). Alternatively, the non-fluent nature of <u>expressive</u> dysphasia (Broca's area). . [Note that *nominal* dysphasia (ability to name shown objects) does not particularly help in distinguishing anatomically between Broca's and Wernickie's area lesions.] <u>Conduction dysphasia</u> – Comprehension intact, and fluent speech, but no ability to repeat a given sentence (lesion in area connecting Wernicke's with Broca's areas)

Dyspraxia. Get patient to demonstrate how he might comb his/her hair, wink or blow a kiss, wave goodbye, put out tongue, imitate speech. Of course, before we can really test for dyspraxia, we must know if there is normal muscle strength and normal comprehension. Agnosia. Recognition of a watch, other objects, by sight, touch, hearing. Differentiation of right from left side (parietal neglect or inattention).. Similar to dyspraxia testing, ie. before we can test for agnosia, we must know whether *peripheral* sensation is intact. Hence these aspects are usually examined later with the spinal motor and spinal sensory systems, during examination of the limbs	Dyspraxia — recognisable particularly in non-dominant hemisphere lesions and need to be brought out by asking the patient to perform complicated movements such as dressing and drawing. But to be demonstrable, there must not be any confusion, dysphasia, motor weakness, or in-coordination; therefore usually test later with spinal motor system (see below). Dyspraxia can sometimes occur with dominant hemisphere lesions, Agnosia - also usually tested later with the spinal sensory system, because we must first know whether ordinary peripheral sensation is intact.
2. CRANIAL NERVES. I. Olfaction	2. CRANIAL NERVES. I. Olfaction Sense of smell.
II. Visual fields and visual attention on confrontation, visual orientation (e.g. recognise whether thumbs up or down). Visual acuity (charts). Also examine fundus with opthalmoscope including vasculature, disc and peripheral retina.	II. Visual fields Recognise vascular narrowing, irregularity, increased light reflex, AV 'nipping', haemorrhages, exudates in hypertension; also papilloedema, macular star in malignant hypertension. Recognise optic atrophy. Other haemorrhages, e.g. blot, dot vitreous haemorrhages in diabetes; sub-hyaloid haemorrhage associated with subarachnoid haemorrhage. Exudates including 'hard' exudates in diabetes; soft 'cotton wool' exudates in hypertension (micro-infarcts). Venous engorgement, particularly in polycythaemia. Venous 'cattle-trucking' in hyperviscosity syndromes. Physiological pitting of disc versus glaucomatous cupping. Various visual field changes and hemianopias, and their anatomical interpretation.
Cranial nerves Ill, IV, VI. External ocular movements (reflex and voluntary), ptosis, nystagmus, exophthalmos, enopthalmus. Pupils, including atrophy, change in size, irregularity, reaction to light (direct and consensual) and accommodation (look for convergence as well as pupillary constriction).	Cranial nerves Ill, IV, VI. Squints - paralytic, concomitant. Ophthalmoplegia, ptosis, Horner's syndrome. Argyll-Robertson pupils, Adie pupil. Nystagmus of different types - see 'Making Physiology work in Clinical diagnosis' – also online at this same website address.
V. Motor - bite and jaw jerk. Sensory — corneal reflex, facial sensation (may be only subjective, e.g. different feeling during shaving etc.) Anterior two-thirds of tongue sensation (not taste).	V. If corneal reflex absent, distinguish whether due to V or VII lesion by asking the patient whether he/she can feel the touch of the cotton wool on the cornea.
VII. Motor - facial movements, including strength of eye closure and lip pursing. Also platysma (everts lower lip). Sensory - taste to anterior 2/3 of tongue.	VII. Paralysis or paresis — distinguish upper motor neurone from lower motor neurone lesion

VIII. Hearing. Use of auroscope to view drum. Tuning fork tests (Rhine's test, Weber's test).	VIII. Distinguish middle ear deafness from nerve deafness
IX, X. Pharyngeal and palatal movement; deglutition, vocalisation; taste and sensation to posterior third of tongue. Gag reflex Involves both (IX) sensory and (X) motor.	IX, X. Recognise bulbar palsy, pseudo-bulbar palsy.
XI. Sternomastoid, trapezius. Test muscle strength of each.	XI. Stemomastoid, trapezius muscle weakness.
XII. Motor innervation of tongue. Observe tongue at rest (atrophy, fasciculation) and during protrusion (deviation, spasticity, tremor); rapid alternating movements (co-ordination); test power with tongue in each cheek in turn	XII. Tongue deviation and its interpretation. Atrophy, spasticity, fasciculation, tremor, rapid alternating movements of protruded tongue. Coating. Distinguish dysarthric from dysphasic speech
3. NECK Know how to test for neck stiffness.	3. NECK Recognise neck stiffness, and detect carotid bruits.
4. LIMBS (a) Spinal motor system Do this completely through as outlined below (comparing each side as you go along), first with upper limbs, then moving to the tower limbs.	4. LIMBS (a) Spinal motor system
(i) *Inspection* - at rest; and under load, e.g. arms outstretched, walking.	*(i) Inspection.* Wasting, fasciculation; involuntary movements, i.e. spasms, tics, convulsions, chorea, athetosis. Tremors, including rest tremors, action or postural tremors, intention tremors, asterixis (metabolic 'flap'). Also observe under load, e.g. patient's ability to maintain posture with eyes closed (upper limbs, arms outstretched; lower limbs — Romberg test). Gait.
(ii) *Palpation*	*Palpation.* Muscle bulk, tenderness. Palpation of nerves.
(iii) *Muscle Tone.* (Important to divert the patient's attention or catch him unawares, otherwise often get voluntary rigidity).	(iii) *Muscle Tone* - types of rigidity/spasticity

(iv) *Power.* Test all muscle groups. Grade any reduced strength, as follows: . 0. – Complete paralysis 1.– Flicker of voluntry contraction 2. – Movt. of limb with gravity eliminated. 3. – Full range movt. of limb against gravity 4. – Movt. of limb against added resistance. 4. – 5. Degree of any reduction of movt against resistance. 5. – Normal power. .	(iv*). Power* - Know important segmental motor nerve root supplies. Upper Limbs. C4. - Abduction of shoulders. C5. – Resisted flexion at elbow. C6. – Resisted dorsiflexion at wrist. C7. - Resisted extension of elbow. T1. – Hand grip Lower limbs L2. – Resisted hip flexion. L3. – Resisted knee extension. L4. – Standing on heels. L5. - Resisted knee extension. S1. – Standing on toes.
(v) *Reflexes,* . Limbs relaxed – distract patient if necessary; reinforcement. Abdominal reflexes, plantar reflexes. . Know how to elicit clonus. .	(v*). Reflexes.* Hyperactive, reduced, pendular (cerebellar). Delayed relaxation phase in myxoedema. Pout reflex in frontal lobe lesions. Finger jerk in upper motor neurone lesions. Know spinal cord segments involved in reflexes as follows. *Deep tendon reflexes* - Biceps (C5, 6). Triceps (C6, 7). Radial (C5, 6). Knee (L3.4). Ankle (L5. Sl). Superficial reflexes - abdominals (T7 -12), Cremasteric (LI, 2), plantars (Sl, 2). *Sphincteric reflexes* - Bulbo-cavernosus (S2-4), anal (S4. 5). Bladder reflexes, potency, continence. *Special reflexes* include Kernig's, reflex leg rigidity. Also know vascular reflexes, viz. the normal blood pressure response to the valsalva manoeuvre, upright posture, mental arithmetic.
(vi) *Test of coordination* including dysdiadokokinesis, finger-thumb apposition, hand and finger tapping. Ataxia including finger-nose test, heel-knee test, Romberg test, fast pointing, heel-toe test. Power and vision must be adequate to interpret these tests. Stand patient up and test base and gait, including heel-toe, and toe-heal walking; hopping on each tiptoe in turn.	*(vi) Co-ordination* - particularly cerebellar inco-ordination. Ataxia - particularly ataxic gait (truncal ataxia). Dyskinesia in Parkinsonism. Hemiplegic gait. Cerebellar ataxia. High-stepping gait with foot-drop. Jerky inco-ordinated gaits with chorea, hemiballismus, hysteria.
(vii) *Dyspraxia* - test where appropriate.	(vii) *Dyspraxia* - parietal lesions. 'Glue-foot' gait – i.e. patient walks as if feet partly glued to floor, and has to concentrate on the act of walking, quite unlike the automatic process it is to us.

(b) Spinal sensory system Light touch and pressure touch. Cutaneous pain (pin, but not so sharp as to draw blood!). Temperature. Pressure pain (tendon squeeze). Joint position sense; vibration sense.	(b) Spinal sensory system Know sensory dermatome.distribution to spinal cord. C2 – jaw; C3 – neck; C4 – shoulders; T4 – nipple line; T10 – umbilicus; L1 – groin. We stand on S1, lie on S2 and sit on S3. Know crossover points within the spinal cord for the different modalities of sensation. N.B. Look for a sensory upper level, espec. with lower limb CNS signs = ? remediable local spinal cord lesion
Cortical sensory recognition Stereognosis (i.e. tests of agnosia including dysgraphaesthesia, astereognosis, left/right perception/attention, two-point discrimination, tactile localisation).	Cortical sensory recognition Recognise agnosia, neglect, and sensory inattention.
GENERAL COMMENT At the end of the routine examination, pause as usual to consider what special things you should be looking for, before leaving the bedside. Then make, quite separately, anatomical and pathological diagnoses, e.g. slow onset of hemiparesis would not suggest 'stroke' from a cerebrovascular cause, but perhaps tumor or some chronic inflammation depending on other features. Once you have made your initial anatomical and pathological diagnoses, ask further questions relating to (dys)function and special pathology, and then to aetiology. Eg. background cigarette smoking, oral contraceptive use, migraine, and alcohol abuse in, say, a young woman presenting with an acute cerebral infarct within the territory of the middle cerebral artery supply, might all be relevant to Aetiiology.	GENERAL COMMENT Be able to diagnose the following lesions: cerebral infarction, haemorrhage, embolism, space occupying lesions; subarachnoid, subdural, extradural haemorrhage. Flaccid and spastic hemiparesis. Parkinson's disease. Brain stem and cranial nerve lesions. Cerebellar lesions. Spinal cord compression. Lower motor neurone versus upper motor neurone paralysis (eg. With 7th cranial nerve). Radiculopathies. Peripheral neuropathy. Peripheral isolated nerve lesions including ulnar, radial, median (including carpal tunnel syndrome) and lateral popliteal nerve palsy.

Joints

1. GENERAL Examine all joints, including neck, spine and other axial joints (costo-chondral, costo-vertebral, sterno-clavicular, acromio-clavicular, vertebral facet joints, sacro-iliac, symphysis pubis) as well as the joints of the extremities. Examine each joint by inspection and palpation, and by putting the limb passively and actively through the range of its various movements (be gentle!). Determine the degree of any *deformity* and, quite separately, the amount of *active inflammation,* because both of these may cause pain and restriction of movement, and treatment is very different	1. GENERAL Examine all joints to determine the *pattern of small and large joint involvement,* because this varies greatly with different conditions and is very helpful in diagnosis.
2. INFLAMMATION Determined on examination by evidence of swelling (including effusions), redness, tenderness, increased warmth, and reduction in active and passive range of movements. Evidence of inflammation on examination can be very subtle, so you should enquire about both general (malaise, lethargy, fever etc.) and local (night pain, rest pain, morning stiffness lasting more than 15 minutes) evidence of inflammation. With more chronic inflammation, synovial swelling &/or thickening may occur, so examine carefully for this.	2. INFLAMMATION Important to distinguish whether pain and limitation of movement are due to inflammation or deformity, as the treatments are so different. Demonstrate effusions, especially in knee (patella tap, 'bulge' sign). Distinguish *synovitis* from other forms of arthritis or joint inflammation, such as articular, bony, or cartilaginous damage.
3. DEFORMITY Determine degree, and look for the presence of joint subluxation /dislocation, as well as limitation of movement from fibrous or even bony ankylosis.	3. DEFORMITY Distinguish limitation of movement due to pain and voluntary muscular spasm from true ankylosis.
4. WASTING of muscles related to joint movement, due to disuse. But, of course, it has other causes.	4. WASTING of muscles. May occur rapidly in any immobilised limb.
5. FUNCTION Assess in relation to patient's daily activities (washing, bathing, eating, sitting down, lying down, walking, housework, shopping, stairs, defaecation, etc.)	5. FUNCTION Observe the patient performing various tasks, preferably in their home situation.

6. ASSOCIATED FEATURES Examine *joints* for cysts, bursae. Examine related *tendons* for evidence of tendonitis, tenosynovitis, synovial fluid or thickening, nodules. Examine *subcutaneous tissue* (nodules). Examine *skin* for rash, vascuiltis. Examine *nails* carefully. Examine *eyes,* including degree of lacrymation; examine sclerae, conjunctivae, iris and anterior chamber, particularly for any evidence of inflammation – associated with some inflammatory joint diseases. Examine *mouth,* especially for ulceration. Examine genitalia, urine.	6. ASSOCIATED PHENOMENA These help to differentiate different forms of arthritis. *Joint signs* - associated cysts, bursae, nodules (e.g. Baker's cyst of knee and Heberden's nodes in osteoarthritis). *Tendon* thickening, including nodules (rheumatoid arthritis); tendon inflammation in the spondyloarthropathies. *Subcutaneous nodules,* particularly over bony prominences, in rheumatoid arthritis; tophi in gout. *Skin* may show evidence of vasculitis related to either rheumatoid arthritis or systemic lupus erythematosis; also evidence of psoriasis. Keratodenna blennorrhagica in Reiter's syndrome. Anaphylactoid purpura (mostly lower limbs) in Henoch-Schonlein purpura. Purpuric skin pustules (target lesions) associated with gonococcal arthritis. *Nails,* vasculitis, psoriasis. *Eyes* - dry eyes in kerato-conjunctivitis sicca (Sjogren's syndrome). Conjunctivitis, iritis, uveitis, associated with various arthritides including ankylosing spondylitis, Reiter's syndrome, Behcet's disease. *Mouth* (mucosal) involvement including ulceration in Stevens-Johnson syndrome (severe erythema multiforme with associated joint inflammation), Reiter's syndrome, Behcet's disease. Gonnococcal pharyngitis. *Genital* involvement (e.g. balanitis in Behcet's disease, Reiter's syndrome). Urethritis in Reiter's syndrome; gonnorrhoea.
GENERAL COMMENT. Distinguish pain due to inflammation from that related to deformity. Always pause after the physical examination of any system, and think what more, in the light of the examination findings, you should go back and enquire about in the history.	GENERAL COMMENT Be able to diagnose the main arthritides, namely rheumatoid arthritis, osteo-arthrosis, rheumatic fever, gout, pseudo-gout, infective arthritides (e.g. gonococcal, HIV). Also the less common, including systemic lupus erythematosis, ankylosing spondylitis, Reiter's syndrome, the enteropathic arthropathies, psoriatic arthropathy, rubella, Behcet's disease, and reactive or post-inflammatory arthritides such as those following enteric infection (e.g. with Salmonella, Shigella, Yersinia, Campylobacter, Chlamydia). Be aware that any sudden flare-up in one joint in a patient with rheumatoid arthritis on steroids may be *infective* in nature (aspirate if slightest doubt).

Haematopoietic / Lymphatic System

1. INSPECTION As usual, take clothes off completely down to briefs. Make sure legs are exposed.	1. INSPECTION Anaemia, plethora, petechiae, eccymoses, bruises, skin infections. Know how to do Hess test in suspected throm-bocytopenia.
2. HANDS	2. HANDS Pallor of palmar creases in anaemia, also koilonychia (in iron deficiency).
3. FACE, including conjunctivae.	3. FACE - pale conjunctivae in anaemia.
4. MOUTH, including throat, tongue, gums, blood vessels.	4. MOUTH - inspect tongue and throat for anaemia; petechiae at junction of hard and soft palate in infectious mononucleosis; petechiae, throat infections in leukaemia, aplastic anaemia. Moniliasis in immunocompromised patient. Herpes Simplex. Blood vessel engorgement in polycythaemia.
5. SKIN. Examine	5. SKIN - infections in agranulocytosis, and immuno-compromised patients; also Herpes zoster in the latter situation. Petechiae in thrombocytopenia. Raynaud's phenomenon in some of the dysgammaglobulinaemias.
6. AREOLAE - inspect.	6. AREOLAE Pale in anaemia (Lowenthal's sign!).
7. LYMPH NODES Neck lymph nodes include supraclavicular, anterior triangle, and posterior triangle. Most nodes in the neck best examined from behind although supraclavicular (especially Virchow's node) should also be examined from anteriorly. Infraclavicular nodes, axillary nodes, epitrochlear nodes. Groins - palpate quite separately for the inguinal and the femoral groups of lymph nodes. With all lymph nodes note size, consistency, tenderness if any, distribution, and whether nodes discrete or matted together.	7. LYMPH NODES Various forms of lymphoma and (lymphatic) leukaemia. Also secondary carcinoma, lymphadenitis.
8. ABDOMEN Feel particularly for liver, spleen. Para-aortic lymph nodes palpable only if large.	8. ABDOMEN Spleen — enlargement in the leukaemla/lymphoma, myeloproliferative and lymphoproliferatlve disorders. Also secondary to liver disease, infections, and in haemolytic states

9. LEGS Examine for petechial spots, rashes, bruises etc.	9. LEGS Petechial spots in anaphylactoid purpura, and some of the hypergammaglobulinaemias. Leg ulcers. Bruising.
10. BONES AND JOINTS Look for bone tenderness, particularly sternal tenderness. Examine joints.	10. BONES AND JOINTS Sternal tenderness may occur in conditions infiltrating bone marrow. Haemarthrosis in haemophilia.
11. OPTIC FUNDI - Examine.	11. OPTIC FUNDI Changes in severe anaemia. Engorged veins with 'cattle-trucking' in hyperviscosity syndromes. Infiltrates and haemorrhages in leukaemia
GENERAL COMMENT This system includes not just formed blood elements (haematopoietic system), but immunological function (lymphatic system) as well. Therefore assess immunological function both on history and examination at the same time. Also we include the reticulo-endothelial system here.	GENERAL COMMENT Recognise the lymphoproliferative and myeloproliferative disorders. Recognise immunological disorders including hypo-gammaglobulinaemias, and dys/hyper-gammaglobulinaemias. Recognise infiltrative conditions such as amyloidosis, reticulo-endothelial system infiltration

Endocrine System

THYROID Know how to examine thyroid gland. First inspection for uniform or irregular enlargement; also ask the patient to take a sip of water, hold it in his mouth, and swallow on command, when a thyroid enlargement should move upwards. Palpate the thyroid gland bi-manually from behind the neck, first generally, then each lobe in turn, which can be made more prominent by pushing the opposite lobe towards the mid-line. In addition, it helps to do bi-manual examination of each lobe from the side with examining hands placed for and aft the sterno-mastoid muscle. Also remember to examine the thyroid isthmus. Thyroid Auscultation Retrosternal percussion Look for pressure symptoms in goitre.	THYROID Differentiate smooth thyroid enlargement of Graves' disease from nodular thyroid goitre. Recognise the signs of hyper-thyroidism including eye signs (lid lag, lid retraction, exophthalmos), tremor, restlessness, appetite increase, weight loss, proximal myopathy, sweating, tachycardia, warm hands, hyperdynamic circulation, thyroid bruit; occasional apathy rather than hyperactivity, pretibial myxoedema. Recognise signs of 'thyroid crisis', with high temperature, ceaseless hyperactivity, and rapid tachycardia – a medical emergency. Be able to diagnose myxoedema clinically from examining face, eyebrows, thinning of hair, coarse skin, deep voice, slow pulse, reduced body temperature, weight gain, weather preference, menstrual change, mental slowing, delayed relaxation phase of tendon jerks and, occasionally, cerebellar ataxia. Recognise difficulty of differentiating thyroid cysts from solid tumours clinically. Recognise clinical characteristics of Hashimoto's disease and/or Riedel's thyroid disease (rare). Thyroid tenderness in sub-acute thyroiditis.
PITUITARY Examine skin, limbs, tongue, jaw, eyes, visual Fields, optic fundi, hair distribution, gonads, blood pressure, urine, body build (height versus span).	PITUITARY Diagnose acromegaly on physical signs including visual fields, optic atrophy, spade-like hands, large feet, lantern jaw, macroglossia, hypertension, glycosuria, and bi-temporal hemianopia from pressure on the optic chiasm. Diagnose other space-occupying lesions (headache, nausea, visual disturbances). Recognise syndrome of inappropriate ADH secretion. Diagnose pan-hypopituitrism - i.e. pituitary dwarf in prepubertal disease. Loss of body hair, hypogonadism, amenorrhoea, change in libido and/or potency, change in hair distribution, galactorrhoea, in adult. Diabetes insipidus in posterior pituitary/hypothalamic lesion.
GONADS Examine testicular size, tenderness. Pelvic examination in the female. Secondary sex characteristics; recognise virilisation in the female which includes not just hirsuitism but deepening of the voice, temporal hair recession, development of male distribution of body hair, clitoral enlargement. (Loss of secondary sex characteristics occurs in adult hypogonadism e.g. from pituitary disease).	GONADS Recognise hypogonadism including that associated with Kleinfelter's syndrome. Boys who have adequate growth hormone and continue to grow in the absence of puberty will develop a eunuchoid habitus with relatively long arms and legs (thus greater span than height). Female virilisation syndromes (ovarian and/or adrenal).

Endocrine System

PARATHYROIDS Examine neck. Know how to look for signs of hypocalcaemia including Trousseau's sign, Chvostek's sign. Examine eyes, particularly towards the periphery of the cornea.	PARATHYROIDS Recognise hypoparathyriodism. Recognise hyperparathyroidism (including band keratopathy near the corneo-scleral junction).
ADRENAL Examine body build, weight, skin, blood pressure, and state of hydration/ECF volume, face, hair distribution, and spine.	ADRENAL Addison's disease — pigmentation, reduced ECF volume with hypotension, lethargy. Cushing syndrome - bruising, thin skin, purple striae, centripetal obesity, buffalo hump over base of neck, glycosuria, virilisation (hirsuitism, clitoral enlargement, male distribution of body hair, deepened voice), moon face, acne, osteo-porosis. Phaeochromocytoma — intermittent hypertension, glycosuria if adrenaline-producing (only adrenal phaeos produce adrenaline).
METABOLIC Skin, particularly below inner angles of eyes (xanthelasma), fundi, urine (glucose, ketones), abdomen, and particularly liver, spleen, kidneys.	METABOLIC Diagnose electrolyte disturbances, hepatic failure renal failure, metabolic acidosis, hyperlipidaemia (xanthelasma, tendon and skin xanthomata, particularly over bony prominences, lipaemia retinalis).
BREASTS Inspection, palpation with the flat of the hand, nipple discharge, axillary lymph nodes.	BREASTS Recognise carcinoma and other lumps.
PANCREAS i.e. Insulin Examine - CNS - urine for glucose, ketones, protein (diabetes mellitus).	PANCREAS Hypoglycaemic attacks— pallor, sweating, tachycardia, confusion. Diabetic presentations - thirst, polyuria, vaginitis, visual change etc. Hyperglycaemic keto-acidosis - confusion, dehydration, Kussmaul's respiration. Hyperosmolar coma - dehydration, osmotic diuresis. Diabetic complications Eyes - cataracts, optic fundi. Peripheral (and autonomic) neuropathy. Vascular disease, both large and small vessel disease
GENETIC	GENETIC Turner's syndrome — short stature, etc. Kleinfelter's syndrome - hypogonadism, gynaecomastia, female body habitus. Down's Syndrome.

Genito-Urinary System

GENITAL1A Always examine genitalia (and lymph nodes) as part of the abdominal examination, and remember to percuss and palpate the bladder as well. Do rectal examination where indicated, examining both the prostate and seminal vesicles	GENITALIA Herniae, epididymo - orchitis. Recognise urinary retention (bladder percussable/palpable or even visible above symphysis pubis). Recognise urinary incontinence, uretheral, meatal obstruction.
GENERAL State of hydration — important to assess in renal disease. Differentiate between extracellular fluid loss (salt and water) and *water* loss (i.e. loss from both extracellular and intracellular compartments). The term 'dehydration' should rightly be reserved for pure water loss, but is often loosely applied to cases of ECF loss (salt and water) as well. With total body *water* loss, the tongue is often dry (although interpretation of this is difficult in mouth breathers); also loss of tissue turgour (best assessed over the cheekbones but even there difficult to interpret in the elderly). Change in bodily weight can be most helpful, especially for changing fluid balance; also history. ECF loss. Salt and water depletion best assessed by history, daily weight, diminished venous pressure (may have to lie patient flat to see it), fall in blood pressure (and increase pulse rate) on *standing;* daily urinary output.	GENERAL Recognise various states of fluid loss particularly sodium and/or water loss and also fluid overload. As with dehydration, ECF overload recognised by weight change and vascular parameters including JVP, as well as evidence of interstitial compartment expansion (dependent oedema). Pure water overload produces cellular over-hydration. Clinically this is manifest mostly in the brain as irritability, confusion and eventually fitting. Severe degrees of water overload produce a fall in plasma sodium (in this context recognise the syndrome of inappropriate ADH effect -SIADH). Recognise nephrotic syndrome; acute renal failure (pre-renal, renal, post-renal). Also recognise signs of chronic impairment of renal function such as anaemia, fluid overload including pulmonary oedema (occasional patients with renal medullary involvement actually get salt loss); also pigmentation, peripheral neuropathy, hypertension, bruising (abnormal platelet function), hypocalcaemia (sometimes with tetany), renal osteodystrophy, signs of hyperkalaemia. Recognise acute glomerulonephritis, pyelonephritis, peri-nephric abscess.
URINE TESTING Inspection Colour - Increased -Decreased -Abnormal Clear or cloudy Specific Gravity test with Multistix. -SG	URINE TESTING Inspection – Dark colour in dehydration, pale in diabetes insipidus. Blood (smoky appearance); urobilinogen (orange, pale-tea coloured). Yellow, often with olive-green appearance when (conjugated) bilirubin appears in the urine - e.g. obstructive jaundice. Absence of bile in the urine in haemolytic jaundice (unconjugated bilirubin is not filtered by glomerulus). Pink urine with porphyrins, heavy concentration of urates, blood, phenolphthalein, beetroot ingestion. Cloudy urine due to phosphates (alkaline pH), urates (in acid or neutral pH, or in refrigerated urine), or suspended cellular elements. Specific gravity Decreased in diabetes insipidus. Increased in diabetes mellitus, dehydration, and after IV Urogram. Fixed (1.010) in renal failure.

Genito-Urinary System

Urinary pH. pH test with Multistix.	Urinary pH. Recognise range of variation in normal. Significance of pH in relation to acid/base balance. Importance of acidifying urine in patients with proteus infection.
Test for Protein (Multistix)	Protein Degree of proteinuria (Multistix are only semi-quantitative. Therefore if more than a trace, do 24-hour urine to quantify). Recognise Bence-Jones protein (light chains) in myeloma (abnormal response to heating urine - may have to concentrate urine to detect).
Test for Glucose (Multistix)	Glucose Diabetes mellitus. Also where low renal threshold for glucose.
Ketones - Multistix adequate in most cases. Older tests such as Rothera rarely needed nowadays.	Ketones Degrees of ketonuria and their significance in relation to starvation, diabetic keto-acidosis. Recognise that diabetic coma can occur without keto-acidosis (non-ketotic hyperosmolar diabetic coma), particularly in the elderly.
Bile constituents Simple tests for urobilinogen/bile in urine (Multistix).	Bile constituents Urobilinogen - trace normally present in urine; excess in haemolytic anaemia, hepatocellular damage. Bilirubin - not normally present in urine. Not present in haemolytic jaundice either. Present in obstructive jaundice or hepatocellular damage (conjugated bilirubin).
Blood It is claimed that Multistix can differentiate between a trace *of haemolysed* blood (uniformly stained strip) and a trace of *non-haemolysed* blood (patchy staining of strip), although in practice this is not always easy.	Blood Differentiate between blood coming from the lower urinary tract, (often admixed with urine) versus upper nephron (often red cell casts, also abnormally shaped cells recognised by phase-contrast microscopy if glomerulus is damaged or inflamed).
Leucocytes: Multistix. Nitrates	Leucocytes. Useful ward test for urinary infection.
Other constituents Can be detected on Multistix.	Other constituents Dipstix (phenistix) can detect phenylketonuria, and also salicylates in cases of suspected analgesic abuse.

Urine deposit Macroscopic appearance. Microscopic examination, of centrifuged deposit. Microscopy is of the utmost importance, and you must know how to do it. Examine for red cells white cells epithelial cells casts: both granular and hyaline phase contrast microscopy to detect abnormally-shaped cells.	Urine deposit Macroscopic appearance — note. Microscopic appearance - recognise different types of casts, abnormal red cells. Special use of phase-contrast microscopy to determine the characteristic abnormally shaped red cells in glomerular damage with the so-called glomerulo-nephritides.

Part IV General Physical Examination

Order of Minimum Routine

The foregoing has concentrated on particular systems, and we now need to consider the order and *minimum* routine of the general examination beyond any one particular system of interest.

1. General Inspection. First observe patient standing and walking where relevant. Then patient stripped to pants, uncovered and lying with chest/neck at angle of about 45°. Stand at the end of bed and observe general demeanour, mood, skin, face, respiration, (including symmetry), neck veins, any pulsation (normal or abnormal), abdomen distension, asymmetry etc.

2. Hands

3. Pulse

4. Blood pressure, sitting and standing.

5. Face

6. Mouth

7. Neck, JVP, carotids, trachea, lymph nodes.

8. Breasts

9. Heart — inspection palpation, percussion; auscultation (also listen in neck and abdomen)

10. Chest — percussion and auscultation of anterior chest, both para-sternally and laterally.

11. Axillary lymph nodes — palpate (bimanual examination may help to trap nodes).

12. Sit patient up,

Listen for aortic diastolic murmur, feel for sternal tenderness, then examine:

(a) *Chest, posteriorly:* observe, percuss and auscultate, particularly over apices, bases.

(b) *Lumbo-sacral area:* oedema; tenderness (including percussion tenderness); limitation of movement.

Neck lymph nodes / thyroid: First from behind, then in front.

13. Lie patient flat: Then examine:

Abdomen — remember to auscultate, especially for epigastric bruits.

Femoral pulses. (including radio-femoral delay, eg. In aortic coarctation).

(c) *Femoral and inguinal* areas, including *lymph nodes.*

(d) Genitalia.

(e) *Popliteal/foot pulses.*

(f) Legs — colour, oedema, varicose veins, calf tenderness, rashes, wasting, abnormal movements.

14. Prop patient up slightly and then examine the nervous/locomotor systems.

Higher cerebral function.

Cranial nerves

Limbs: Upper limbs first then lower. Compare both sides as you go along. *Inspection and palpation* First, *(including joints);* examine at rest and during movement.

Note any tenderness, swelling, fasciculation, wasting, tremor, or involuntary movements. Then check tone, power, reflexes, co-ordination and sensation (touch, pinprick, vibration, position).

15. Sit patient up, with legs dangling over side of bed, and examine optic fundi.

16. Get patient to walk if not done already; hop on each tip-toe; heel-toe etc. .

17. Examine particular areas after performing manoeuvres that precipitate symptoms, in appropriate cases (see also below).

18. Make sure you have examined urine and taken temperature, and done rectal/pelvic examination where necessary.

Comments.

At end of whole examination, pause and think of what more you should examine in the light of the history and findings so far. Particularly if the symptoms are episodic, and episodes are precipitated by accessible factors, put the patient under the appropriate load and re-examine — e.g.

- the complaint of 'giddiness' on neck movements should be further examined by moving the patient's neck appropriately and observing for nystagmus (including Hallpike manoeuvre);
- obscure shortness of breath on exertion should be further investigated by re-examining the patient (particularly the cardiovascular and respiratory systems) after exercise
- a complaint of staggering should be examined by observing gait (obvious but easy to forget when you get caught up too much in the routine).

You must be able to do your *routine examination efficiently, accurately* and *thoroughly.* It must become like driving a car, i.e. sufficiently routine so that you are not too pre-occupied with its performance to miss additional clues in the individual case, or to be thrown off the order of your routine by following them up.

There are three aspects of the general examination which students tend to forget and should be thought about at the end of each examination. They are:
Temperature, urine examination and *blood pressure.*

Make sure that you have determined all of these before leaving the bedside.

Remember:

1. Remove all clothing except underpants/briefs for initial inspection.
2. Position patient correctly.

3.Start with an initial inspection from end of the bed.

4. Approach your patient as a person, introduce yourself, get his confidence, and don't hurt him/her.

5. Approach each step first with an open, then with a more closed mind, i.e. pause at the end of each routine step and ask what special aspects you might like to do now in the light of clues from history and examination so far.

6. Before you leave the patient, ask yourself whether you do have enough information to make a complete anatomical, pathological, functional and aetiological diagnoses (clinical), and if not whether you should ask further *questions,* particularly about the aetiological background (e.g. 'risk-factors' for ischaemia heart disease).

7. If there are *multiple symptoms/signs* remember the following in diagnosis:

   (a) Always *try to see the condition first as one primary underlying problem* with secondary symptoms/signs occurring elsewhere as a consequence (e.g. liver disease with secondary splenomegaly, gynaecomastia, ascites, parotid enlargement, spider naevi, testicular atrophy, jaundice etc.).This is the important principal of Occam's razor!

   (b) If you are having trouble seeing which (symptom) is cause and which is effect, in synthesising your diagnosis, go back to the history and find out which *came first in time,* e.g. myocardial ischaemia and atrial fibrillation could be related either way around, so find out whether the patient noticed chest pain or palpitation first.

   (c) Always be on the look out for especially unusual presentation *of reversible diagnoses.* They are, after all, what matters.

Part V Clinical Examinations

Comment on approach to observed clinical examinations

Get on with things — examine each part, reach your conclusions and *then* give your findings (precisely) at the *end of each phase.* Don't waste time. Don't talk about what you are going to do or about what you might do! Do each *segment* within each system; silently, and then give your findings.

For example: In the CVS, give findings at each stage after completing your examination of the hands, the blood pressure, the neck (JVP), the heart (including auscultation of all areas), the lungs, the abdomen, the peripheral pulses, the optic fundi. By and large the examiners want to *watch* your examination performance in each segment, and are only interested in hearing your conclusions at the *end of each* stage, not in your speculations about what you might do! Examine the part correctly and efficiently, give your findings accurately and precisely and interpret them with decision.

On management*:*

1. First make sure diagnosis is correct, and that you have considered and excluded important *reversible* conditions (by investigational tests if necessary).

2. *Problem list.* Draw one up in complicated cases (often multiple problems in the elderly).

3. *Define objectives* of investigation and treatment: e.g.

   (a) are you looking for *cure or palliation.*

   (b) investigation and treatment of *functional manifestations.*

   (c) investigation and treatment of *underlying pathology.*

   (d) investigation and treatment of underlying *anatomical* problem (e.g. coronary angiography and coronary artery bypass surgery, where indicated).

   (e) investigation and treatment of underlying aetiological *'risk factors'* for further recurrence (e.g. smoking and heart attack).

   (f). primary *preventative treatment,* (e.g. hypertension treatment mainly for prevention of heart attack/stroke. (Therefore treat other vascular 'risk factors' as well i.e. smoking, obesity etc.).

Conclusion.

The details of knowledge, clinical performance and diagnosis are now up to you. This booklet is meant merely to provide guidelines for allowing you to see the wood through the trees.

Get on now and practise it.

Part VI. Conclusions

I leave you with a few thoughts and quotations of wisdom in Medicine.

On history-taking

Did you use words the patient could not understand?

Was the patient's real problem concealed amongst your insignificant questions?

'The best kind of history-taking is at the same time the best kind of psychotherapy.' (Ericson).

On examination

Do not touch the patient at first - merely state what you see; this cultivates your powers of observation (after William Osler).

The gentle touch of a good physical examination is at the same time an important means of reassurance and therefore healing.

On diagnosis

'Never lose a holy curiosity.' (Albert Einstein).

"The great questions are those an intelligent child asks, and getting no answers stops asking." (George Wald).

Common sense is not common.' (Voltaire).

When you hear hoof beats, think first of horses, not zebras.

'Every psychoneurotic ultimately dies of organic disease.'

On investigation

No patient was ever cured by a laboratory determination.

The more questionable the indications for requesting a laboratory test, the greater the problems the answer will cause,

Or: 'Do a silly test and you get a silly answer.' (Merskey's rule)

On management

Littman's rule — the longer it takes for a condition to develop, the more time you should take to return the patient toward normal.

Life is not a drug-dependent state.

Always remember the old Egyptian proverb: 'Health is a crown on a well man's head, but no-one can see it but a sick man'.

On Information

'To study the phenomena of disease without books is to sail an uncharted sea, while to study books without patients is not to go to sea at all.' (William Osler).

....Where is the wisdom we have lost in knowledge?

Where is the knowledge we have lost in information? (T.S Eliot).

Textbooks of a previous generation were as large as the textbooks of today, but contained a different body of misinformation!

'Statistics are like a bikini. What they reveal is suggestive; what they conceal is vital . . .' (A. Koestler).

On Common Sense!

Finally, always remember Loeb's laws of medicine,

If what you are doing is working, keep doing it.

If what you are doing is not working, stop doing it.

If you don't know what to do, don't do anything.

Above all, never let a surgeon get your patient!

I wish you not luck, but understanding.

G.W. Boyd 2002